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Frontispiece.

ROBBEN ISLAND LEPER AND LUNATIC ASYLUMS, SOUTH AFRICA.

A

HANDBOOK

ON

LEPROSY

BY

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ROBBEN ISLAND LEPER AND LUNATIC ASYLUMS, CAPE COLONY, SOUTH AFRICA

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TO

HIS EXCELLENCY HENRY BROUGHAM LOCH,

BARON DRYLAW,

KNIGHT GRAND CROSS OF THE MOST HONOURABLE ORDER OF THE BATH,

KNIGHT GRAND CROSS OF THE MOST DISTINGUISHED ORDER OF

ST. MICHAEL AND ST. GEORGE,

LATE GOVERNOR AND COMMANDER-IN-CHIEF OF HER MAJESTY'S

COLONY OF THE CAPE OF GOOD HOPE IN SOUTH AFRICA, AND OF THE

TERRITORIES AND DEPENDENCIES THEREOF,

AND HER MAJESTY'S HIGH COMMISSIONER FOR SOUTH AFRICA, ETC. ETC.,

WHOSE KINDLY INTEREST IN THE WELFARE OF THE LEPERS ON

ROBBEN ISLAND DID MUCH TO LIGHTEN THEIR BURDEN,

This Handbook

IS RESPECTFULLY DEDICATED BY

THE AUTHOR.

PREFACE.

As chief and medical superintendent of the Robben Island Infirmary, which in addition to its lunatic asylums, has one of the largest leper settlements in the world, I had special opportunities of studying leprosy.

In 1892 the Leprosy Repression Act was put into force in the Cape Colony, and a large number of lepers was sent to the island from the various districts of the Cape Colony, and also from some of the neighbouring States.

I was struck with the fact that many patients were sent to the island as lepers who were not suffering from the disease.

As all medical men have not equal opportunities of becoming practically acquainted with the different forms of leprosy, it occurred to me that if a small handbook, well illustrated with photographs of leper patients in all stages of the disease, were written and presented to the public, it would not only be of assistance to the medical men who have to deal with the cases, but would be of interest to the public generally, especially at a time when the leprosy question is engaging the attention of all classes to such a degree. It is with the humble endeavour to supply these much-felt wants that I have written this handbook.

S. P. IMPEY.

January, 1896.

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A HANDBOOK ON LEPROSY.

INTRODUCTION.

THIS handbook has been prepared in the hope of assisting those who have not had an opportunity of becoming well acquainted with leprosy, in recognising the disease, and appreciating its nature.

The book does not claim to be anything but a practical aid to those in want of assistance.

Many other writers have entered more fully and minutely into the various branches of the subject, but it has been my endeavour to place before my readers, in as concise a form as possible, all that is at present known concerning it. I have consequently purposely refrained from mentioning the various theories that have from time to time gained credence as to the cause of the disease, more especially as it is now almost universally acknowledged that leprosy is due to the presence in the system of the bacillus lepræ. I have for the same reason omitted to discuss the question of heredity, more so as [illegible] are that there are now very few supporters of [illegible] to its causation.

I have, by a careful selection of photographs, endeavoured to illustrate, in a practical manner, the most important physical signs of leprosy, in its various forms and stages, as I know from experience that one ocular demonstration is more instructive than pages of the most descriptive writing.

I have mentioned a fourth form of leprosy—a form which is not generally recognised, and this may give rise to adverse criticism; but, from a practical point of view, I feel that I am more than justified in doing so, as syphilitic leprosy has more marked distinguishing characters than any of the other forms of the disease, and I believe it is by not recognising this form that so much confusion has arisen in previous descriptions of leprosy.

My ideas as to the cure of the tubercular form of leprosy during its earlier stages, by means of erysipelas, may also be adversely criticised; but all observers must acknowledge, and therefore agree with me, that acute intercurrent affections have a beneficial effect on the local signs of tubercular leprosy. Knowing this, I am led to believe that in these affections we have a means, if properly used, which must lead to its cure.

The photomicrograph, which I have prepared of the bacilli in the cutis, illustrates very well the points which I wish to elucidate.

In discussing the history of the disease I have confined my remarks to that of South Africa, as its records elsewhere have been fully written by other writers.

CHAPTER I.

HISTORY.

1. Native Traditions—Dutch East Indies.

In the History of the Cape Colony no mention is made of leprosy until the middle of the last century, so it may be reasonably concluded that until then the disease was either unknown or not very prevalent in the European settlement.

The traditions of the natives indicate the existence of the disease amongst the Hottentot tribes prior to the advent of the white man; but from whence it came, and to what extent it existed, it is impossible to form an opinion.

Leprosy was probably introduced into the Dutch settlement from the Dutch East Indies, where the Europeans obtained their servants.

I shall as briefly as possible mention a few of the most important incidents in connection with the history of leprosy in the different South African States, and show to what extent it at present prevails in these countries.

2. Cape Colony.

In 1756 two European farmers residing near Stellenbosch were reported to the Government to be suffering

from leprosy. They with their families were isolated on their own farms, and apparently nothing further was done with regard to the disease until 1817, by which time it had increased to such an extent that it was deemed necessary to make some provision for the isolation of patients.

"Hemel-en-Aarde" (Heaven on Earth), an isolated spot amongst the Caledon mountains, was set apart as a leper asylum for the colony, and to it patients from all parts were drafted.

During the twenty-eight years this asylum was in existence it gave shelter to upwards of 400 lepers. In 1845 the patients who were then resident in Hemel-en-Aarde were transferred to Robben Island, which has since then been used as the leper settlement of the colony. From October, 1845, until the end of December, 1894, there were 1,771 leper patients admitted into the Robben Island Leper Asylum, making, with the 400 admitted into Hemel-en-Aarde settlement, a total of 2,171 lepers who were treated in the leper asylums of the Cape Colony since they were first opened in 1817; but as only paupers and voluntary patients were admitted into the asylums, prior to the promulgation of the Leprosy Repression Act in 1892, the number of lepers actually admitted probably represents only a small proportion of the lepers who existed in the colony.

At present, as far as can be ascertained, there are about 150 lepers still at large in the European districts of the Cape Colony, and there are 450 colonial lepers detained in the Robben Island Asylum.

3. Orange Free State.

In 1835, for political reasons, the "Emigrant Boers" left the Cape Colony, and settled in what is now the Orange Free State. They probably took leper servants with them, and thus introduced the disease into the new colony; but, as will be seen, the natives who inhabited the western portions of the new state probably had leprosy amongst them before the arrival of the farmers from the Cape Colony.

So far as can be ascertained there are at present about 150 Free State lepers, of whom, however, many are confined in the Robben Island Asylum.

4. Transvaal Republic.

Some of the emigrant farmers from the Cape Colony passed through the Free State, and crossing the Vaal river, settled in the Transvaal, where they founded the present Republic. They also probably introduced leprosy into this new country.

A small hospital has long been erected for the accommodation of about twenty lepers, near Pretoria, the chief town of the Republic, but as there has been no law making segregation of lepers compulsory there, it is impossible to say to what extent the disease exists amongst the people, especially as it is chiefly confined to the natives who are living in a more or less savage state; but, according to a report framed by a commission which was appointed by the Raad to inquire into the matter of

leprosy in the Transvaal, the disease is decidedly on the increase in that country, and the settlement which a few months ago contained only twenty lepers now gives accommodation to upwards of sixty. The Commission recommends that a law should be made enforcing the segregation of lepers.

5. Natal.

The disease in this British colony is almost wholly confined to the natives, who state that it was introduced among them about 1843.

The story, which has received the most credence amongst the natives themselves, and which is probably the correct one, is as follows:—

In 1840 two Natal boys left their homes to look for work in the Cape Colony, and, whilst temporarily employed in Grahamstown, they lived with a coloured woman, who was a leper. They returned to Natal in 1843, and shortly afterwards became lepers.

It was by these two men that leprosy is supposed to have been introduced into Natal, in which colony there are at present upwards of 200 cases. The Natal Government is now taking steps to isolate their leper patients.

6. Basutoland.

Until 1835, the Basutos were a very exclusive race, and held no intercourse with strangers, but at this time the country was thrown open, and a number of Bushmen, driven by the European settlers from the Free State,

made homes for themselves in Basutoland, and introduced leprosy amongst the people. Leprosy is at present known in Basutoland as the Bushmen's disease. There are at this time upwards of 250 lepers in this country.

7. Bechuanaland.

Bechuanaland is inhabited by a large native population, among whom leprosy is not very prevalent, and there are probably not more than a dozen lepers in the country. The disease was introduced into Bechuanaland by the wandering Bushmen from the Free State.

8. Griqualand East.

In 1863, the Griquas, a Hottentot tribe, amongst whom the disease was rife, were moved by the Govèrnment from the western districts of the Orange Free State to No-Mansland, now called Griqualand East, into which country they introduced the disease.

There are at present about 130 lepers in this native territory belonging to the Cape Colony.

9. Transkeian Territories.

These territories adjoin Griqualand East, and leprosy was probably introduced into them by the Griquas, as it was only a few years after the arrival of the strangers that leprosy was first discovered amongst the aborigines. So far as I can gather there are about 400 lepers in these lands.

10. Pondoland.

According to the Pondo laws a leper is not allowed to live in the country; he may leave it, or he must die. There are consequently no lepers in this country, which has been so recently annexed to the Cape Colony.

11. Zululand and Swaziland.

Leprosy exists in both these native territories, but to what extent is not known.

From information obtained, I gather that there are at present upwards of 2,000 lepers in the various above-mentioned South African States.

CHAPTER II.

DISTRIBUTION.

LEPROSY, as already indicated, exists to some extent throughout the whole of South Africa, but it is most prevalent in the native territories, and in those districts of the Cape Colony in which there is a dense population, especially of coloured people; it is also more frequently met with in the agricultural than in the pastoral districts.

The following table shows the various districts of the Cape Colony, the number of lepers from each district admitted into the Robben Island Asylum since 1890, the total population of each district, the number of European and coloured people respectively, and the proportion of lepers per 10,000 of the inhabitants in each district:—

District.	No. of Lepers.	Total Population.	European.	Coloured.	Prop. per 10,000.
Aberdeen ..	1	6,435	3,106	3,329	1·53
Albany	22	23,377	9,391	13,986	9·41
Albert	4	16,649	8,193	8,456	2·40
Alexandria ..	6	10,005	2,417	7,588	5·99
Aliwal North ..	14	9,963	4,661	5,302	14·05
Barkly West ..	10	17,487	3,404	14,083	5·72
Bathurst ..	1	9,197	1,833	7,364	1·08
Beaufort West ..	5	9,239	3,875	5,364	5·41
Bedford ..	17	11,682	2,301	9,381	14·55

District.	No. of Lepers.	Total Population.	European.	Coloured.	Prop. per 10,000.
Bredasdorp ..	0	6,607	3,271	3,336	*nil*
Caledon ..	33	12,192	5,821	6,371	27·06
Calvinia ..	2	12,255	5,050	7,205	1·63
Cape	123	97,283	48,544	48,739	12·64
Carnarvon ..	0	9,132	3,733	5,399	*nil*
Cathcart ..	3	6,891	2,119	4,772	4·35
Ceres	13	5,973	2,488	3,485	21·76
Clanwilliam ..	10	11,568	4,473	7,095	8·64
Colesberg ..	7	8,288	3,464	4,824	8·44
Cradock ..	12	15,049	6,517	8,532	7·97
East London ..	9	21,538	7,197	14,341	4·17
Fort Beaufort ..	19	14,675	3,135	11,540	12·94
Fraserberg ..	1	6,907	3,528	3,379	1·44
George	4	10,076	4,947	5,129	3·96
Graaff Reinet ..	14	16,378	6,202	10,176	8·54
Hanover ..	0	4,301	1,854	2,447	*nil*
Hay	7	8,508	3,526	4,982	8·22
Herbert ..	1	9,074	2,434	6,640	1·10
Herschel ..	9	25,059	193	24,866	3·59
Hope Town ..	2	6,500	3,038	3,462	3·07
Humansdorp ..	15	11,846	4,130	7,716	12·66
Jansenville ..	0	9,370	4,170	5,200	*nil*
Kimberley ..	34	48,306	20,306	28,000	7·03
King Williams-town	33	86,983	8,605	78,378	3·79
Knysna ..	3	6,930	3,710	3,220	4·32
Komgha ..	3	6,941	1,345	5,596	4·32
Ladismith ..	1	6,704	3,652	3,052	1·49
Malmesbury ..	45	23,328	10,120	13,208	19·20
Middleberg ..	4	9,689	4,042	5,647	1·12
Mossel Bay ..	3	7,286	3,445	3,841	4·11
Murraysberg ..	5	4,453	1,498	2,955	11·22
Namaqualand ..	1	16,945	3,718	13,227	·59
Oudtshoorn ..	15	23,670	11,576	12,094	6·33
Paarl	28	21,403	8,266	13,137	13·08
Peddie	2	16,525	1,458	15,067	1·21
Phillipstown ..	2	6,846	3,214	3,632	2·92

District.	No. of Lepers.	Total Population.	European.	Coloured.	Prop. per 10,000.
Piquetberg ..	14	11,587	6,515	5,072	12·08
Port Elizabeth ..	10	25,408	13,939	11,469	3·93
Prieska	1	4,302	2,018	2,282	2·32
Prince Albert ..	0	7,046	3,716	3,330	*nil*
Queenstown ..	17	42,895	6,458	36,437	3·96
Richmond ..	5	7,246	2,868	4,378	6·90
Riversdale ..	9	11,366	6,203	5,163	7·91
Robertson ..	5	11,348	6,019	5,329	4·40
Somerset East ..	12	19,007	6,740	12,267	6·31
Stellenbosch ..	43	12,780	4,420	8,360	33·64
Steynsberg ..	0	7,052	2,676	4,376	*nil*
Stockenstroom ..	12	7,776	1,660	6,116	15·43
Stutterheim ..	7	8,651	1,967	6,684	8·00
Sutherland ..	0	4,012	2,191	1,821	*nil*
Swellendam ..	3	11,256	5,585	5,671	2·66
Tarka	0	7,443	3,149	4,294	*nil*
Tulbagh ..	0	5,654	1,865	3,789	*nil*
Uitenhage ..	7	20,947	7,185	13,762	3·34
Uniondale ..	0	8,415	3,927	4,488	*nil*
Victoria East ..	1	8,875	1,242	7,633	1·12
Victoria West ..	1	7,220	3,406	3,814	1·38
Willowmore ..	0	9,036	4,342	4,694	*nil*
Wodehouse ..	4	37,149	9,491	27,658	1·07
Worcester ..	6	12,616	5,086	7,530	4·75

The following table shows the area of the above named districts of Cape Colony in square miles, the total population of each district, the number of people to each square mile, and the number of lepers admitted into the Robben Island Infirmary since 1890 from each of the districts:—

District.	Area.	Population.	Per sq. mile.	Lepers.
Aberdeen	2,645	6,435	2·4	1
Albany—C	1,685	23,377	13·8	22
Albert	2,660	16,649	6·2	4
Alexandria—C	947	10,005	10·5	6

District.	Area.	Population.	Per sq. mile.	Lepers.
Aliwal North	1,305	9,963	7·6	14
Barkly West—A ..	4,024	17,487	4·3	10
Bathurst	573	9,197	16	1
Beaufort West	6,374	9,239	1·4	5
Bedford—A	1,225	11,682	9·5	17
Bredasdorp	1,577	6,607	4·19	—
Caledon	1,772	12,192	6·9	33
Calvinia	23,784	12,255	·5	2
Cape—B	663	97,283	146	123
Carnarvon	12,069	9,132	·7	—
Cathcart	995	6,891	6·9	3
Ceres—B..	3,871	5,973	1·5	13
Clanwilliam	6,046	11,568	1·9	10
Colesberg..	2,394	8,288	3·5	7
Cradock	2,973	15,049	5	12
East London—C.. ..	682	21,538	31·7	9
Fort Beaufort—A ..	860	14,675	17	19
Fraserberg	9,950	6,907	·7	1
George—B	979	10,076	10·3	4
Graaff Reinet—A ..	2,692	16,378	6	14
Hanover	2,105	4,301	2	-
Hay	6,646	8,508	1·2	7
Herbert	2,763	9,074	3·2	1
Herschel—C	660	25,059	37·9	9
Hopetown	4,302	6,500	1·5	2
Humansdorp—A ..	1,950	11,846	6	15
Jansenville	1,923	9,370	4·8	—
Kimberley	1,764	48,306	27·3	34
King Williamstown—C..	1,327	86,983	65	33
Knysna	810	6,930	8·4	3
Komgha—C	546	6,941	12·7	3
Ladismith	1,254	6,704	5·3	1
Malmesbury—B.. ..	2,329	23,328	10	45
Middleberg	2,222	9,689	4·3	4
Mossel Bay	707	7,286	10·3	3
Murraysberg	2,035	4,453	2·1	5
Namaqualand	18,462	16,945	·9	1
Oudtshoorn	1,653	23,670	14·4	15

District.	Area.	Population.	Per sq. mile.	Lepers.
Paarl—B.	610	21,403	35	28
Peddie—C	657	16,525	25·1	2
Phillipstown	2,695	6,846	2·5	2
Piquetberg—B	1,733	11,587	6·6	14
Port Elizabeth	176	25,408	144	10
Prieska	5,284	4,302	·8	1
Prince Albert	4,293	7,046	1·6	—
Queenstown—C	2,194	42,895	20	17
Richmond	4,430	7,246	1·6	5
Riversdale	1,712	11,366	6·6	9
Robertson	1,526	11,348	7·4	5
Somerset East	3,052	19,007	6·2	12
Stellenbosch—B	318	12,780	40	43
Steynsberg	1,113	7,052	6·3	—
Stockenstroom—A . .	314	7,776	24·7	12
Stutterheim—C	670	8,651	12·9	7
Sutherland	4,808	4,012	·8	—
Swellendam	2,362	11,256	4·7	3
Tarka	1,427	7,743	5·4	—
Tulbagh—B	373	5,654	15·1	—
Uitenhage—C	2,973	20,947	7	7
Uniondale	1,680	8,415	5	—
Victoria East—C . .	330	8,875	26·8	1
Victoria West	4,873	7,220	1·4	1
Willowmore	3,498	9,036	2·5	—
Wodehouse—C	3,664	37,149	10·1	4
Worcester—B	2,623	12,616	4·8	6

The districts marked "A" are occupied by a large number of half-castes of the Hottentot tribe; those districts marked "B" are largely populated by coloured Cape people of mixed extraction; the districts marked "C" are occupied by pure bred natives. Kimberley has in its native miners a large coloured population from all parts of South Africa. Caledon is the district in which the old Leper Asylum was situated, and at Graaff Reinet

and Uitenhage lazarettoes were erected for the reception of lepers prior to their transmission to Hemel-en-Aarde. Many lepers belonging to the districts inhabited by the pure bred natives are still at large.

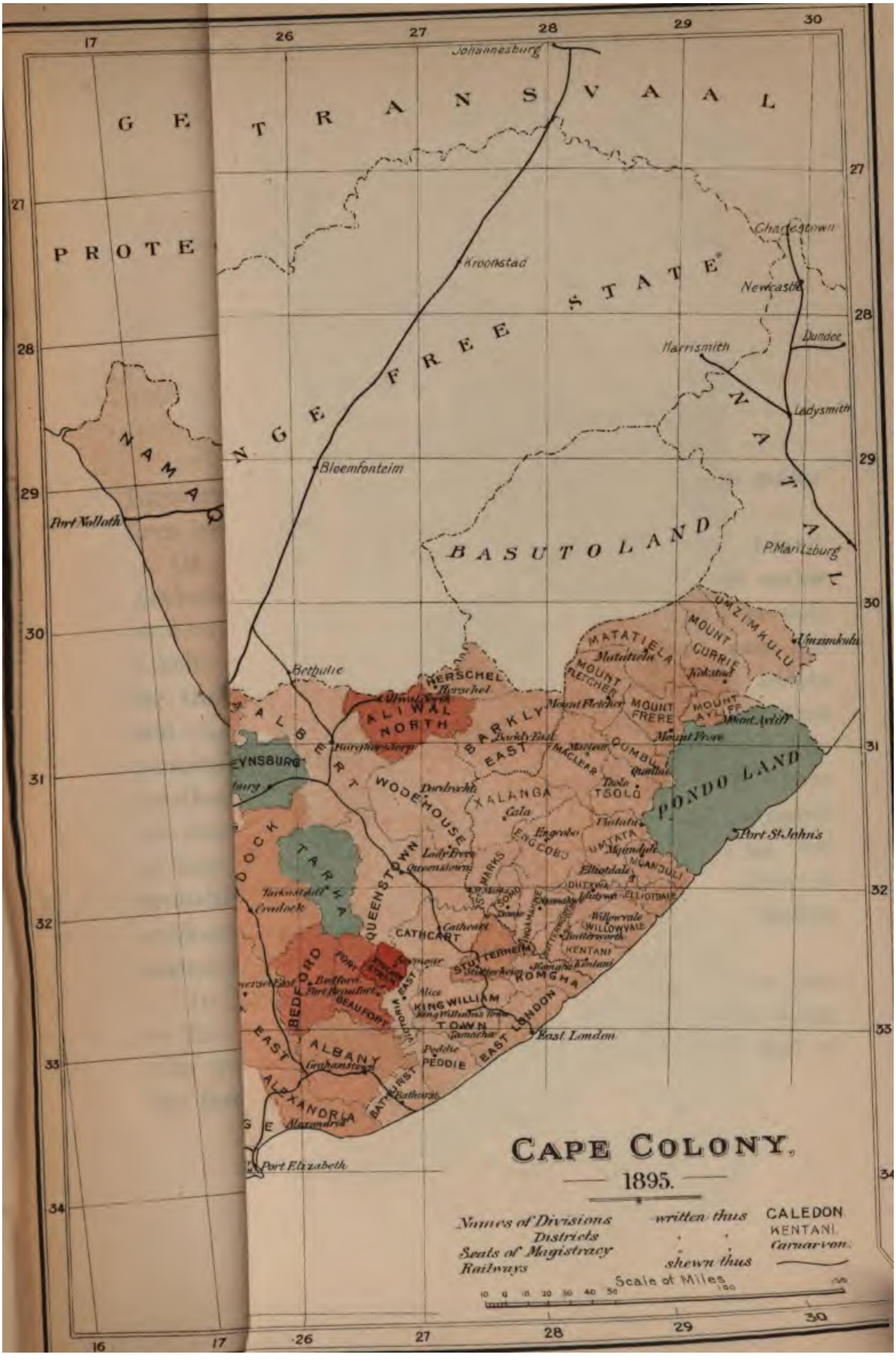
From these tables it would appear that leprosy exists chiefly in those districts in which there is a more or less dense population, and especially of coloured people of the Hottentot tribe or half-castes.

The sparsely populated districts of the colony (which are also the pastoral districts) are comparatively free from leprosy, and the prevalence of the disease appears to have no reference to the situation of the district as regards the sea-board.

The map which I have prepared shows that there are as many lepers in the inland districts as there are in those near the coast—due regard being taken of the population of the districts.

The countries uncoloured, are beyond the limits of the Cape Colony. The districts marked blue have sent no lepers to Robben Island since 1890.

The districts coloured red are those in which leprosy exists, the number of lepers being in proportion to the density of the colouring. The old leper colony of Hemel-en-Aarde accounts for a very large number of the lepers at present existing in the colony.


CAPE COLONY.
1895.
Names of Divisions written thus CALEDON.
Districts KENTANI.
Seats of Magistracy shewn thus Carnarvon.
Railways
Scale of Miles
TRANSVAAL
ORANGE FREE STATE
BASUTOLAND
NATAL
PONDO LAND
Johannesburg
Kroonstad
Bloemfontein
Charlestown
Newcastle
Dundee
Harrismith
Ladysmith
P.Maritzburg
Bethulie
Port Nolloth
Umzimkulu
Port St. Johns
East London
Port Elizabeth
HERSCHEL
ALIWAL NORTH
ALBERT
BARKLY EAST
WODEHOUSE
XALANGA
Cala
QUEENSTOWN
TARKA
CATHCART
BEDFORD
FORT BEAUFORT
ALBANY
Grahamstown
ALEXANDRIA
BATHURST
PEDDIE
EAST LONDON
KING WILLIAM TOWN
KOMGHA
KENTANI
WILLOWVALE
TSOLO
UMTATA
ENGCOBO
MATATIELE
MOUNT CURRIE
UMZIMKULU
MOUNT FLETCHER
MOUNT FRERE
MOUNT AYLIFF
MACLEAR
QUMBU

CHAPTER III.

SEX.

LEPROSY in South Africa occurs more frequently in males than in females.

Of the patients admitted into the Robben Island Asylum for Lepers, since 1845 there were 1,296 males as compared with 475 females. This difference is not, I believe, due to any predilection which the disease has for the male sex, but to the difference in the habits and occupation of the two sexes; for it would appear that persons who are exposed to the vicissitudes of the weather, and who are engaged in hard manual labour are more liable to contract the disease than those engaged in less trying and arduous pursuits. This may be, and probably is, due to the fact that the former class of workers is more liable to injury, and thus to have broken surfaces and open wounds.

In all countries, whatever proportion the males bear to the females in the population, the number of males who suffer from leprosy is always in excess of that of the females.

CHAPTER IV.

AGE.

LEPROSY attacks persons of all ages. The youngest person in the Robben Island Asylum, suffering from leprosy, contracted the disease at the age of three years; but there are patients in the asylum who contracted it when upwards of eighty years of age.

The following table shows the age of the patients who have been admitted into the asylum since 1891, in quinquennial periods:—

Age of Patients	Number of Patients admitted in each period.			
	To 1891.	In 1892.	In 1893.	In 1894.
1— 5 years	—	1	1	—
5—10 „	7	4	6	4
10—15 „	36	25	15	12
15—20 „	45	40	47	15
20—25 „	48	52	26	6
25—30 „	48	40	33	13
30—35 „	47	40	29	11
35—40 „	25	20	26	12
40—45 „	37	28	20	13
45—50 „	18	26	14	14
50—55 „	22	24	6	10
55—60 „	17	10	13	6
60—65 „	13	17	9	4
65—70 „	1	5	3	—
70—75 „	6	2	1	—
75—80 „	7	2	1	2
80—85 „	—	2		
90—95 „	1			
Totals ..	378	338	250	122 = 1,088 patients.

Average age of	378	patients	admitted	up to the end of		1891,	33½ years.
„	„	„	338	„	„	in the year 1892,	34 „
„	„	„	250	„	„	„ 1893,	31 „
„	„	„	122	„	„	„ 1894,	34½ „

Total ... 1,088 patients, and their average age 33½ years.

The average age of all the patients admitted into the Robben Island Asylum during these years, at the time of admission, is, therefore, 33½ years.

This table shows the average age of the patients at the time of their admission, but not that at which the disease was contracted; to arrive at this it will be necessary to deduct about 4½ years from the average age at time of admission.

Leprosy is, as a rule, not very marked before it has existed for about three years, so that very few patients are admitted into a hospital before the third year of its duration, and the majority of the patients die within six years of contracting the disease, so that the greater number would be admitted some time between the third and the sixth year of its continuance; it is for this reason, that, I think, if we deduct 4½ years from the average age at the time of admission, we should arrive at the period when the disease was contracted, which would accordingly be about 29 years.

CHAPTER V.

FORM.

LEPROSY in South Africa occurs in four forms: the tubercular, see Plate III.; the anæsthetic, see Plate XVI.; the mixed, see Plate XXVI.; and the syphilitic, see Plate XXXI.

Without at present going into details, it may be stated generally, that in the tubercular form of leprosy the skin of the face, hands, and feet, is affected with tubercles; in the anæsthetic form the nerves of the extremities and of the face are diseased, causing atrophy and ulceration of the tissues, paralysis, and contraction, and more or less amputation of the digits; in the mixed form of leprosy the tubercular and the anæsthetic forms are combined to a variable extent in the same patient; in the syphilitic form the symptoms of leprosy are modified by the presence in the system of the syphilitic poison.

The lepra mutilans mentioned by some writers is nothing more than anæsthetic, or, as it is sometimes very properly named, nerve leprosy, in an advanced stage and severe form. Anæsthetic leprosy does mutilate the extremities in some cases to a terrible degree, as may be seen by referring to Plates XVIII. and XX.

CHAPTER VI.

RELATIVE FREQUENCY.

In South Africa, as in all warm and temperate climates, most of the patients suffer from the anæsthetic form of the disease.

Of the 703 patients admitted into the asylum here since the beginning of 1890 there were 369 anæsthetic cases, 238 tubercular, 81 mixed, and 15 syphilitic, making a percentage of 52·48 of anæsthetic, 33·86 of tubercular, 11·53 of mixed, and 2·13 per cent. of syphilitic cases.

The proportion of tubercular cases amongst the females is slightly higher than amongst the males, and, curiously enough, almost every European female at present in the asylum is suffering from tubercular leprosy.

In cold climates the proportion of tubercular cases is always much higher. In Norway, for instance, according to Danielssen and Boeck, 51·6 per cent. of the cases of leprosy are tubercular, while only 33·3 are anæsthetic, and 15·1 are mixed.

This is, in my opinion, not a mere coincidence, but is due to the peculiar nature of the disease, as I shall point out later on.

CHAPTER VII.

CAUSE.

It is now almost universally acknowledged that leprosy is due to the presence in the system of the bacillus lepræ. If the bacillus attacks the skin, we have tubercular leprosy; if it attacks the nerves, we have anæsthetic leprosy; if it attacks both the skin and the nerves, we have mixed leprosy; and if the bacillus attacks a patient suffering from syphilis, or if syphilis is contracted by a patient suffering from leprosy, we have syphilitic leprosy.

Why the bacillus in some cases attacks the skin and leaves the nerves, and in other cases attacks the nerves and leaves the skin alone, is difficult to say.

I am inclined to think that either there are two very similar but not identical bacilli in leprosy, one of which has a predilection for the skin, and the other for the nerves, or that in pure anæsthetic leprosy the poison secreted by the bacilli acts in a more powerful manner upon the nerves, because the system of the patient is peculiarly susceptible to the effects of the ptomaine.

In a tubercular case the bacilli are more numerous, but the poison they secrete does not injure the patient to any great degree; but in anæsthetic leprosy, though the number of bacilli is very much less, yet the patient is so susceptible to the poison that it sets up violent inflam-

mation of the nerves, while in the tubercular cases the nerves are not visibly affected.

I believe that in a case of pure anæsthetic leprosy the nerves are affected without the bacilli themselves necessarily entering the nerves—that is to say, I believe the poison, and not the bacilli, causes the disease in the nerves; otherwise, how is it that in so many cases of anæsthetic leprosy it is impossible to find the bacilli in the nerves? Some observers have found the bacilli in the nerves of anæsthetic patients, but I have not done so. May this not be due to the fact that they have mistaken mixed for anæsthetic leprosy, especially as in many cases the tubercular symptoms and signs are almost wholly masked by the predominating anæsthetic symptoms? This is, however, only a matter of opinion, and I may have failed in my examination of the nerves through faulty manipulation. Still the facts remain that in one form of leprosy the nerves are destroyed, and in the other the skin is affected and the nerves escape; and also, that in the form in which there are very few bacilli the nerves are attacked, whereas in the form in which the bacilli produce large areas of thickened skin the nerves are not much, if at all, diseased. The question is, Is the inflammation in the nerves due to the poison, or to the bacilli themselves?

If the neuritis is caused by the poison acting on a peculiarly susceptible person, then the difference in the mode of action of one bacillus in the two forms of the disease can be understood, but if this is not the explanation, then the only other one that can account for the two forms satisfactorily is, that there are two different, though perhaps almost identical, bacilli.

It will not be necessary for me to mention all the

theories that have from time to time been propounded to account for leprosy, as they have all been exploded by the discovery of the bacillus lepræ by Dr. Hansen. All are willing to acknowledge that this bacillus is the cause of leprosy, the only points that are still matters for discussion are, where does the bacillus come from? and how does it enter the body?

Even these are now almost settled, for it has been satisfactorily proved, and is now almost universally acknowledged and agreed, that the bacillus cannot live outside the human body; or, in other words, that it comes from the human body, and that it enters a new abode by broken surfaces, and no medium as yet discovered has proved suitable for the cultivation of the bacillus lepræ.

CHAPTER VIII.

BACILLUS.

THE bacillus occurs in enormous numbers in the skin of the affected parts in tubercular leprosy (see Plate X.), and it occurs to a less extent in the nerves in anæsthetic leprosy.

It may be readily demonstrated in the following ways:—

1. IN THE SERUM.

Clamp a tubercle, or tie an elastic band around it until it becomes anæmic, then prick it with a needle or sharp lancet. Place the drop of clear fluid which exudes on a cover glass, let it dry, and when dry, pass it rapidly through the flame of a spirit lamp. This not only fixes the film firmly, but assists the process of staining. In passing it through the flame be careful to hold the prepared side upwards. It is now ready for staining. Place a few drops of an aqueous solution of fuchsine on the film, and, after a minute or two, wash the stain off in water, and examine the specimen under a microscope with a power of 300 diameters or more, when the bacilli will be seen as delicate pink rods. A five per cent. solution of

carbolic acid added to the fuchsine appears to assist the process of staining—probably the acid acts as a mordant. The bacilli may be seen all over the field in countless numbers, some apparently in cells, others free among the corpuscles.

When examined under an instrument of high power the stained bacilli sometimes appear to be knotted, there may only be two knots, or there may be more, but usually there are not more than four in a bacillus.

Some observers have, I think, erroneously concluded that these knots represent spores. I am of opinion, however, that they are produced in the process of staining by the action of the staining fluid upon the delicate material with which the external membrane of the bacillus is filled. This albuminous matter becomes hardened and contracted, and is thus broken up into two or more parts; the spaces between the broken parts do not take on the stain, but the albuminous matter does, hence the appearance of knots or spores.

Opposite each space the cell-wall also seems to become contracted, so that the appearance presented by the stained portion of the bacillus might most readily be mistaken for spores.

A bacillus with four knots is equal in length to about half the diameter of a red blood-corpuscle.

2. In the Tissues.

(*a*) Place a properly cut section of a tubercle on a cover glass, put thereon a drop of acetic acid, and examine with a low-power microscope without staining, and the

masses of bacilli may be readily seen as brown irregular spots on a clear ground. This is a most excellent way of making a diagnosis in a disputed case of leprosy, as the brown masses are, in my opinion, pathognomonic of the disease.

(*b*) To stain the bacilli in the tissues, place the section in a solution of fuchsine in aniline water for some hours. Remove it carefully and dip it for a second into a 33 per cent. solution of hydrochloric acid. This will, if properly done, remove the stain from everything but the bacilli. Wash the section in water and examine, or, if you wish, stain the tissues with methylene blue, the bacilli will then appear as pink rods on a blue ground.

The lepræ bacillus may be stained in many other ways, for which I must refer you to text books on the subject of staining, but for all practical purposes the methods I have mentioned will suffice.

CHAPTER IX.

COMMUNICABILITY.

It is now, I think, generally acknowledged that leprosy is a contagious disease, that it is not infectious, and that heredity has little, if anything, to do with its spread.

There are at present in the Robben Island Asylum 266 leper parents who have had 951 children; of these children twenty-three became lepers, that is less than 3 per cent. of the whole.

There are at the present time 520 lepers in the asylum, of whom 475 were born of healthy parents. Of the remaining forty-five cases the father alone was affected in twenty-five, the mother alone in sixteen, and the father and mother were both diseased in four only.

This certainly is a strong argument against heredity, especially as the forty-five cases may have, and did probably, contract the disease by coming into contact with leprous discharges; for who is so likely to contract a contagious disease as the child of a parent suffering from such a scourge?

In dealing with a loathsome disease like leprosy, one has to be very cautious in accepting the statements of the patients concerning their family history, as the tendency is to deny the taint; but, nevertheless, the figures above

quoted not only indicate that heredity can have very little to do with the spread of leprosy, but they would almost lead one to think that the children of lepers are to some extent "protected" against it.

No external influences, climatic, telluric, or meteorological, have any appreciable effect on the spread of leprosy; but, as I have already stated, it appears to be more prevalent in the agricultural than the pastoral districts of the Cape Colony, which may be accounted for, I think, by the peculiar nature of the work on an agricultural farm—work which necessitates close and intimate intercourse of individuals, and the free interchange of tools, implements, &c.

Leprosy is spread by actual contact of broken surfaces, but of course it may be produced by deliberate or accidental inoculation, or by the bacilli being carried from a leprous sore to a healthy wound by some intermediate body, such as the handle of a knife or instrument, a pipe, or even by flies or other insects, or by vegetables and fruits previously handled by lepers with open wounds. The bacillus appears, however, to be a weak one, and unless the medium into which it is introduced is in every way suitable for its reception and growth, it will die.

The system of the recipient of the bacillus must, therefore, be specially prepared for the microbe, prepared either by poverty, dirt, unhealthy surroundings, or by constitutional or acquired debility. In such a system the bacillus finds a suitable medium, but even then it does not at once grow and flourish. It remains quiescent for a time, and appears to be, as it were, dormant, until it is stirred into activity by some exciting cause.

If you ask a leper how he contracted the disease, he will almost invariably reply that it was due to a cold.

Now, though this is not strictly correct, yet there is, I believe, a great deal of truth and force in this prevalent idea, for I am of opinion that cold is the exciting cause of leprosy.

The bacillus lies inactive in the system until it is excited into action by the body being subjected to severe cold.

CHAPTER X.

INCUBATION PERIOD.

After the introduction of the bacillus into the system, and until it makes its presence known, it probably circulates slowly through the lymphatic system safely protected from harm in a leucocyte.

This incubation period of leprosy is of variable extent, as it is terminated, I believe, by accidental causes.

It is not impossible for the bacillus of leprosy to be for years in the systems of the persons who show no signs of the disease, and I think it is quite possible, if the body is well protected against the exciting cause of leprosy, to prevent the bacillus from doing harm, for it is only when it commences to grow and multiply that the system suffers. I have known a case in which the bacillus has begun to grow, and thereby produce symptoms of leprosy within three months of its introduction into the system.

The difficulty of obtaining thoroughly reliable data holds good of all diseases in which the latent period is lengthened, and leprosy is no exception, if any reliance can be placed upon the statements of the patients them-

selves and upon reported cases, which go to show that in many instances the incubation stage is what I might term unduly protracted. However, from the study of a large number of cases, and careful inquiry into the antecedents, I am led to believe that in the majority this period does not exceed two years.

CHAPTER XI.

PATHOLOGY AND MORBID ANATOMY.

In order to be able to comprehend and fully appreciate the clinical history and symptoms of any disease, it is necessary to know its pathology and the condition of the parts affected. I have, therefore, thought it advisable to reverse the usual order of things, and to demonstrate the morbid anatomy of leprosy before discussing its most important signs and symptoms.

In order to be able to elucidate the pathology of leprosy, I shall describe in detail the probable life-history of a bacillus from the time it enters the body till it has accomplished its deadly work. In order to do so, I shall deal, as it were, with one bacillus, or with one colony of bacilli, whereas, in reality, many millions of bacilli and generations of microbes co-operate in producing such terrible results in the human body (see Plate X.).

It has not yet been ascertained how long a bacillus lives in the system, though we know that, under certain conditions and circumstances, a microbe may cause disease many years after it has left, or been removed from, the medium in which it thrived; but as we have neither been able to cultivate the bacillus of leprosy, nor to produce the disease artificially, even in the human body, very

little is known of its capabilities, and it is by no means certain that it can exist for a moment in any other abode.

The bacillus enters the frame by an open wound. Here it encounters the defenders of the body, the leucocytes, which are ever collected round a vulnerable part to protect the system from invasion, and it attacks them.

If the leucocytes prevail, the bacillus is destroyed; if the bacillus is the victor, it enters one of the leucocytes and there takes up its abode. Safe in this cell, it probably courses slowly through the lymphatic system and spaces of the body, though how it travels is not actually known.

Thus it passes through the system of a patient and visits almost every part of the body; but as it is not found in the blood, it either does not enter the general circulation, or, what is more likely, is probably destroyed as soon as it gets into the veins by the chemical action which goes on in the lungs.

A patient with this bacillus in the system is then exposed to a severe cold, which produces partial stagnation of the blood in the capillaries of the skin.

The parts most likely to suffer from sudden changes in the weather are the face, hands, and feet, not only because they are more exposed than the rest of the body, but because they are the parts farthest removed from the centre of circulation, and are thus the least able to resist injury or repel attack. The leucocytes, with their bacilli, congregate at these congested spots, and, escaping from the vessels, accumulate in the lymphatic and perivascular spaces of the part. The vitality of the skin is further lowered by the partial stagnation of the blood, thus setting up a condition favourable to the growth of the bacilli, which at once begin to develop and multiply by fission.

If the bacillus thus retained is in the skin, tubercular leprosy follows; if it is caught in the nerves, the result is anæsthetic leprosy.

From this point the history of the two forms of the disease begins to diverge, and it will be necessary for me to deal with each separately.

(*a*) Tubercular Leprosy.

The skin in man is mapped out by almost invisible depressions into innumerable "islands." The skin of the patient, represented in Plate IX., through being much swollen and infiltrated by the deposit of tuberculous material, shows these markings and islands much magnified.

Each "island" has its own complete system of blood-supply, its own arterial twigs, and its own veins and lymphatics, which form a minute network of looped vessels. The apex of this loop is directed towards the cuticle, and the base towards the deeper structures of the skin.

It is in or about the apex of this capillary loop that the bacilli form their first colony; they do not invade the papillary layer of the skin to any great extent. From the summit of the bend, as a centre, the colony increases by spreading at the circumference, until the whole island is crowded with bacilli and their products.

The centre of a colony is more or less compact, and is composed almost entirely of granular matter, probably disintegrated bacilli, but the periphery contains a number of leucocytes with bacilli.

The bacilli grow in the lymphatic and perivascular spaces, and not in the veins and arterioles.

It is probable that as soon as the leucocyte, with its occupants, escapes from the vein, as a result of the congestion, the bacilli begin to develop and multiply. The first effect of their multiplication is the secretion of a poison, which acts as a solvent, and thus destroys the cell inclosing them, giving the bacilli freedom of action to continue their work.

They not only grow most rapidly, but each bacillus, as it is separated from its parent by fission, secretes a gummy matter, which binds it to its fellows, until the whole colony forms a more or less compact mass. The number of bacilli to be found in a tubercle thus formed is very great—greater than in any other known disease, as may be well seen in Plate X., where the colonies may be observed in various stages of development.

The original island is soon filled with masses of bacilli, and their products and its neighbours are encroached upon until the whole thickness of the skin of the part affected is infiltrated with the leprous material. Even in the most advanced cases of leprosy, however, the original boundary between two distinct and neighbouring settlements of bacilli may be seen in a microscopic section of a tubercle, the various colonies or centres of growth being separated from each other by more or less dense bands of fibrous tissue. The bacilli, in addition to secreting the gummy matter, which binds the individuals together, appear to form a sort of soluble poison, which enters the general circulation and produces certain results, which I shall fully describe when discussing the symptoms of leprosy; but this noxious ptomaine has a local effect on the tissues, which very materially affects the progress of the disease.

The poison, the exact nature of which has not been

defined, acts as an irritant solvent, for it not only destroys the tissues of the part affected, but sets up violent inflammation in the tubercle, effecting the partial destruction of the tissues, and the formation of bands of inflammatory material in and around the colony which created it.

The bacilli do not pass beyond the limits of the true skin at this stage, nor do they encroach upon the cuticle, but in some cases the inflammation they produce is so severe that there is an effusion of fluid between this and the skin, with the formation of the so-called bullæ.

The bacilli are chiefly confined to the denser portions of the true skin, next the epidermis; but they do invade the less dense layers of the cutis beneath, by passing along the lymphatic spaces, which are more or less distended with their products. These masses may be readily seen with a low-power microscope in a section of a tubercle, in the form of round tubular projections, passing down into the areolar tissues of the skin.

By accumulating in the perivascular spaces, the bacilli compress and partially close the channels, thus causing an obstruction to the free flow of the blood, with consequent swelling and turgescence of the part. In time, however, the capillaries are more or less obliterated by the foreign mass; the hair bulbs are encroached upon and destroyed by the pressure; the hair consequently dies and is extruded; the sweat ducts are obliterated; and the secretion of sweat over the part affected either ceases completely or is very much diminished. The sebaceous glands are at first pressed upon, and there is an increased secretion of oily matter, which, escaping by the very patent orifices, lubricates the skin, making it appear shining and greasy;

but the glands are soon also destroyed, and the skin becomes hard and dry.

In my opinion the bacilli do not invade the ducts, and I believe that whilst the cuticle is unbroken they cannot escape, and that a leper is, therefore, safe whilst his epidermis is intact. This is a most important fact in dealing with patients suffering from the tubercular form of the disease.

The bacilli increase in numbers most rapidly, and, by doing so, gradually obliterate the veins and arterioles in the tubercles, and the tissues, being deprived of their blood-supply, consequently become necrosed and slough (see Plate IV.).

The ulcer thus formed presents an opening through which the bacilli and their products are discharged in countless numbers; hence it is that the ulcers of tubercular leprosy are so prolific of danger to the public, and I am of opinion that it is from the ulcers of this form of the disease that leprosy is spread by inoculation.

How many of the bacilli so discharged are alive it is impossible to say, for we have not yet discovered a medium outside the human body in which the leprous bacillus will grow, and, with the exception of perhaps one very doubtful instance, the deliberate introduction of the bacillus into the system of a healthy person has failed to produce the disease; so that it is impossible to prove that any of the bacilli which escape from a leprous ulcer are in a fit state to carry on their mission. Probably only a few of them are alive and, therefore, dangerous.

There are, in my opinion, grave doubts as to the existence of the so-called leper cell.

I believe that the bacillus in the incubation period of its existence takes up its abode in a leucocyte, but that these cells are destroyed as soon as the bacilli commence to grow and multiply, and that, while the bacillus is there, it is harmless; but, as soon as it leaves the cell, it commences its deadly work.

The so-called cells are, in my opinion, the masses of bacilli bound together by the homogeneous gummy matter which they secrete while multiplying. These cells and the bacilli are well seen in Plate X.

It would appear that each bacillus, as it is separated from the parent, secretes a small amount of this gum, which binds it to its fellows; when this process is repeated many times a mass is produced, in which the bacilli may be seen lying in all manner of positions, some deeply embedded in the mass or only partly so, others again are on its surface, and in a stained specimen as many bacilli are seen scattered generally about the tissues as are found in the masses or cells. I cannot accept the suggestion that the cells are ruptured in the preparation of the section or specimen, as an explanation of the presence of the bacilli outside of the cells, for if the serum be carefully extracted from a tubercle or ulcer as many bacilli are floating in the plasma as are found in the leucocytes, and here the prick of a needle could hardly have ruptured the cells.

I am inclined to believe that the bacilli leave the leucocytes as soon as they reach a spot in the system which is suitable to their growth, and I am of opinion that the cell is not necessary to their existence, and I believe that the bodies called leper-cells are zooglia and nothing more.

The masses are of all sizes and shapes, though they generally assume a more or less circular form. It may be that the free bacilli are alive, and that those in the masses are dead or devoid of life. This opinion is strengthened by the appearance of the bacilli in these cells or masses; they appear smaller and less defined, as though they were, to a certain extent, disintegrated, and in a stained specimen they look more like grains than rods.

Some observers state that they have seen movements in the bacilli, but I have not been able to verify this.

When the contents of a tubercle are thus evacuated the ulcer soon heals and becomes covered with a scab, and a beneficial effect is produced on the adjoining parts by the discharge relieving the tension to some extent.

The leprous growth presses upon the terminal twigs of the cutaneous nerves which pass into the tubercle, and thus, in a certain degree, paralyses them, with the result that in an old tubercle, which has become hardened by much infiltration, the cuticle over it becomes more or less anæsthetised.

In such a case the pressure is from without, and it is not due to interstitial neuritis as in anæsthetic leprosy, for, if the pressure is removed from the nerve, its function is restored. In anæsthetic leprosy the pressure is within, and the nerve-fibres are destroyed so that the function of the nerve is never regained.

The bacilli do not multiply at a uniform rate, but they appear to grow at irregular periods. Why this should be so is difficult to say, but I believe that colds and draughts, by acting upon the skin, cause changes in the circulation of the part favourable to the growth of the bacilli; but, be this as it may, the fact remains that at certain times they

multiply rapidly, whilst, again, at others they are more or less quiescent. With each exacerbation the tubercles become swollen, tense, and turgid, and there is more or less general pyrexia, which I believe is produced by the ptomaine secreted by the bacilli during this stage of activity.

Each of these congestive attacks has a marked permanent effect on the tubercles. The number of bacilli is enormously increased, and the density of the growth also by the deposit of inflammatory lymph: these two actions going on simultaneously must lead either to the death of the part by necrosis through overcrowding, or arrest of the growth through want of space.

I have frequently noticed that old-standing tubercles become contracted, hard and fibroid, which state is due, I believe, to these repeated attacks of congestion, giving the appearance, to a certain extent, of the bacilli working out their own destruction. The inflammation which they produce causes a fibroid degeneration of the tissues which, in its turn, destroys the microbes.

This aspect of the question is one deserving of every consideration, as, I believe, it is by accelerating this fibroid degeneration of the tubercles, or, by producing it artificially, that the bacilli in tubercular leprosy will be destroyed and leprosy cured.

The tubercles may form on any part of the body, but in a large majority of cases they are confined to the face and extremities.

When the disease is well advanced the bacilli invade the mucous membrane of the mouth, throat, eyes, and nostrils. Beginning at the lips, they gradually spread backwards until the mucous membrane of the mouth, the

tongue, hard and soft palates, the fauces and upper part of the larynx, are studded with small and somewhat pale and flattened tubercles. As the disease progresses in the throat the breathing becomes more and more affected, the voice harsh and croaking, and the tubercular swelling of the Schneiderian membrane gives the voice a nasal twang.

The tubercles vary very considerably in size in the different parts of the body, those in the mucous membranes are uniformly smaller than those in the skin, but they pass through the same stages wherever situated. Those in the mucous membranes are paler in colour, and, if anything, are less prone to ulceration than those in the cutis.

The discharges from the ulcers in the mucous membranes have a peculiar sweet, but very fetid, odour, very characteristic of the disease.

The tubercles which form in the eyes are small and vascular, being first developed in the conjunctiva of the lower eyelid, whence they spread until the whole eye is disorganised (see Plate XXVIII.).

When the bacillus first takes up its abode in the skin, it secretes a poison which enters the general circulation, and has a marked effect on the terminal branches of the cutaneous nerves.

The primal effect of this poison on the nerves is that they become partially paralysed, causing vasomotor paralysis, and the formation of erythematous patches.

These patches are similar to those of ordinary erythema, and are accompanied by a considerable amount of irritation, discomfort, and pain. Most of them soon fade and disappear, leaving no trace behind; a few, however, become stationary, and in them are formed

tubercles. These erythematous patches must not be confounded with those of anæsthetic leprosy, which are due to disorganisation of the cutaneous nerves and not to mere temporary paralysis.

I have not been able to discover the bacilli in the internal organs of the body, but other observers have found them in the liver, spleen, ovaries, and kidneys.

Many of the internal organs do undoubtedly become diseased in tubercular leprosy, but I do not think the diseases thus produced are necessarily leprotic, but are the result of septic poisoning and excessive ulceration.

(*b*) Anæsthetic Leprosy.

In this form of the disease the bacilli do not attack the skin but only the nerves.

The nerves of the extremities and of the face are those chiefly affected, but others are also sometimes attacked. The cutaneous nerves are invariably injured by the poison secreted, but I doubt if the bacilli actually penetrate these in all cases.

The sequence of events in this form of leprosy is not known exactly, but the following is probably the life-history of a bacillus in anæsthetic leprosy.

I am inclined to believe that the microbe does not always enter the nerves, but that the destruction of these is brought about by the poison which the bacilli secrete. This poison has undoubtedly a most pernicious effect on them, as may be seen by the effect it has on the cutaneous nerves in the first stage of the disease, which are more or less destroyed by its influence; but whether the

bacilli themselves actually enter there or not is of little consequence, as the results are the same—the nerves are not destroyed by the bacilli, but by their products.

That portion of a nerve which from its position or surroundings is most liable to injury, or is most exposed to external influences and impressions, is the part which is the first to become affected by inflammation, as for instance, the ulnar at the elbow, the median at the wrist, and the peroneal at the knee.

Interstitial neuritis is set up in the exposed part of the nerve, the diseased portion becoming swollen and soft. There may be one of these swellings in the course of a nerve, or there may be many, but as a rule each spot of inflammation is distinct, and it may be readily detected during life as a nodular enlargement or thickening of the nerve.

Its whole thickness, too, may be affected, or only a portion of it—that is, all the fibres of the nerve at the swollen part may be inflamed, or only a few of them. Those not affected may be perfectly sound, and, as may be imagined, the severity of the symptoms and the amount of destruction of the tissues is in proportion to the amount of destruction of the nerve-fibres. Should all the fibres be destroyed in a mixed nerve there follows complete loss of nerve-power, and the tissues normally supplied by it are disorganised. If, however, only a few of the fibres are destroyed, then the effect on the tissues may be only very insignificant; for instance, there may be only a slight numbness of the part, or the whole extremity may be disorganised and lost. Between these two extremes there are, as may be supposed, innumerable gradations, according to the amount of disorganisation of nerve-fibres.

The neuritis at first causes irritation of the part supplied by the inflamed nerve, which is accompanied by shooting pains extending from the congested spot to the extremity of the affected limb. A shock to the nerve at this stage is very painful, and any pressure thereon gives rise to considerable discomfort. A jar in walking is often enough to send a shock, like that from an electric battery, through the body.

As the irritation continues the muscles supplied by the nerve become involved, and the fingers flexed. The small finger is the first to become contracted (see Plate XII.), but it is soon followed in turn by the ring, middle, and index fingers, the thumb being usually the last to suffer (see Plates XIV. to XVII.).

I have noticed that this contraction is due to paralysis, and it is not due, as some suppose, to shortening of the ligaments.

The muscles now become atrophied and wasted, and the adipose tissue disappears from the part affected, so that the hands, in addition to being contracted, become emaciated and thin, and the bones appear to be abnormally prominent (see Plate XVII.).

If the nerves are only slightly affected the disease may not extend beyond a slight contraction of the fingers, with partial paralysis of the part in question, and slight atrophy of the tissues; but if severely injured necrosis of the bones follows. The first sign of a diseased bone is a swollen, tense, and glistening condition of the finger or toe accompanied by considerable pyrexia. In a few days the swelling shows signs of suppuration, becoming soft and fluctuating, and if left to itself soon bursts and discharges a considerable quantity of sanguineous pus—the

ugly ragged wound thus formed extending down to the bone, which has become necrosed (see Plate XIII.).

If this bone is removed by operation the wound heals readily, but if left to nature it discharges a considerable quantity of matter, and remains open until the bone is loosened and extruded, when it at once heals.

In this manner one bone after another becomes necrosed and lost, until a point is reached where the parts have sufficient nerve-power left to keep them alive, though perhaps not in a perfect state of health. At this point—or as it may be called, line of demarcation—the ulceration ceases. As I have before mentioned, if the nerves are only slightly diseased these local symptoms may not, for instance, proceed beyond a slight contraction and numbness of the fingers, but with a greater amount of destruction of the nerves the whole of the digits (see Plate XVIII.), the hands, and even part of the radius and ulna may be lost (see Plate XX.).

The corresponding bones in the upper and lower extremities are usually affected to a similar extent, so that a description of the disease, as it affects the arms and hands, will suffice for the legs and feet.

The disease is nearly always bilateral, and the nerves are usually affected to the same extent on both sides of the body, but occasionally one side is more affected than the other. In this form of leprosy the skin of the extremities is not primarily affected, saving that it becomes wasted and anæsthetised, but as the parts are devoid of feeling, and have, moreover, a lowered vitality, the slightest injury to the part causes disorganisation of the tissues, so that the patients have generally a considerable number of wounds about the hands and feet. I believe that the

bones become primarily diseased, and by thus becoming necrosed give rise to abscesses and ulcers. I am opposed to the opinion that necrosis starts on the surface and extends inwards to the bone, and I think that with the exception of the accidental wounds above referred to, if it were not for the necrosed bone which it incloses, but which has to pass through it to be extruded, the skin would remain intact. I am strengthened in this view by the fact that in some cases the bones are removed, not by necrosis, but by absorption, and then there is no ulceration of the skin (see Plate XX.).

If the ulcers are not caused by the bone, then how is it that there are none when the bones are absorbed, though the tissues are equally atrophied and anæsthetised to the same extent as when the bones become diseased? This leads me to discuss the perforating ulcer of leprosy, about which so much has been written.

If the bone which has become diseased is a phalanx it is readily cast off and the wound heals, but if the bone is firmly wedged in between other bones—as for instance, a metacarpal or carpal bone—it cannot be readily removed by nature even though it be extensively diseased, so that it remains *in situ*, in some cases for many years: a sinus leading to this dead bone is called a perforating ulcer.

To prove that I am correct in my opinion as to the nature of these ulcers, I have frequently removed the dead bone at the bottom of one of many years' standing, with the result that the wound has healed at once.

The perforating ulcer is, therefore, I consider, only a sign of deep-seated necrosis due to the defective innervation of the part; in some cases the part to become

necrosed is not a bone, but a large tendon or bundle of fibrous tissue. As the ulcer is due to necrosis the question remains, Is the disease to be considered active until the necrosed tissue is removed? This is a point I wish particularly to accentuate, as I believe there are many patients at present confined in leper asylums in whom the disease has long ceased to exist. A young man, as the result of periostitis, had a large sequestrum in the shaft of his femur, which, after ten years, I removed; during this long period the patient suffered much pain and discomfort, for the sinus leading to the dead bone was not only continually discharging, but the irritation set up by the sequestrum had caused considerable contraction of the knee-joint. I removed the bone, and within a very short period the patient had recovered the use of his leg and the wound healed. No one would be justified in considering that the youth had been suffering from periostitis all these years, and yet he would have suffered all his life probably if the bone had not been removed.

In leprosy the destruction of the nerves causes the death, so to speak, of the bones. The bacilli may, and probably do, disappear from the system, for they cannot be detected in the nerves of an old-standing case of anæsthetic leprosy, but the dead bone remains firmly impacted for years. Is it right to consider such a patient as suffering from leprosy just because the bone is still *in situ?* Remove the dead bone and the wound heals at once, and if the ulcer is the only sign that remains of the disease, then by the removal of the dead bone causing the sore, the disease must be cured; but as I believe that the dead bone may exist when the bacilli have long

ceased to be present in the body, it is not leprosy that is cured, but its effects.

"Once a leper, always a leper" has so long been impressed upon the minds of people that they can hardly be made to believe anything to the contrary, but I do consider that leprosy can be, and in many cases is, cured, and that, therefore, the old saying is incorrect. I am of opinion that tubercular leprosy may be cured in its early stages by appropriate treatment, that anæsthetic leprosy is often cured spontaneously, and that the existence of a perforating ulcer is no indication that the disease is not cured.

The destruction of the nerves often leads to terrible deformities, but deformities are by no means a sign of disease. A patient has small-pox, which pits his face with indelible marks, yet we should not be justified in considering that until the marks disappear, the patient is suffering from small-pox. In the same way we are not justified in considering a patient to be suffering from leprosy because the marks remain. I contend that when the active symptoms disappear, even though there may be a perforating ulcer, the patient is cured of leprosy, and the disease permanently arrested.

Even in these arrested or cured cases the patients are continually injuring themselves, as the extremities are anæsthetic; but these wounds are not leprotic and soon heal.

In an advanced case of anæsthetic leprosy the nerve is almost completely destroyed, the originally swollen and soft nerve becomes indurated and contracted through interstitial inflammation, until, in some cases, it is represented by a fibrous filament almost devoid of nerve-tissue. All those of the extremities are liable to become disorganised, but I have not met with any cases in which

the nerve-trunks have become affected; the ulnar, the radial, the median, and occasionally the musculo-spiral nerves in the arm, and the peroneal and tibial in the leg, with the facial branches of the seventh and fifth nerves are those most usually diseased.

In anæsthetic leprosy the disease is almost invariably bilateral, and it is generally of equal severity on both sides of the body (see Plate XIII.). This has often led me to think that the destruction of the nerves in anæsthetic leprosy is not due so much to the bacilli themselves as to the poison which they secrete.

A person becomes affected with leprosy, the bacillus gains a footing in the body and secretes a poison which enters the circulation, and sets up inflammation in the nerves which become disorganised. In tubercular leprosy the nerves are affected by this poison, and erythematous spots are formed in the skin; in anæsthetic leprosy the cutaneous nerves are affected in the same way and with the same results, but in the latter form of the disease there appears to be a stronger dose of the poison, so that instead of the cutaneous nerves recovering their power again, they become more or less completely destroyed, and the patches remain permanently. The difference may be, as I have already pointed out, due to a peculiar susceptibility of the patient to the effects of the virus.

The skin becomes atrophied through loss of nerve-power, and the hairs thin and bleached, but they do not fall as in tubercular leprosy; the sebaceous and sweat glands atrophy and shrink, so that the amount of their secretions is much diminished, and the colouring matter of the skin more or less removed from the Malpighian layer (see Plates XXIII. and XXIV).

I believe that these effects are due to the poison and not directly to the bacilli.

In a person peculiarly susceptible to the action of this poison, it may readily happen that its effects may extend deeper and the larger nerves become affected. Its effect may be increased by the idiosyncrasy of the patient, or the poison in these anæsthetic cases may be introduced into the system in a more powerful dose; but be this as it may, I am of opinion that the virus has more to do with the destruction of the nerves than the bacilli themselves. If this theory be correct, it may explain how it is that in so many cases of anæsthetic leprosy it is impossible to find the bacilli in the nerves.

After the most careful examination of numerous nerves I have not been able to find the bacilli, though they are so readily demonstrated in tubercular leprosy.

The constant irritation, and the great drain on the system, caused by the ulcers, in this form of leprosy lead to anæmia and great general debility, and they also set up amyloid degeneration of the internal organs, such as the liver and kidneys, and it is generally these secondary affections that kill the patient.

In both forms of leprosy the lymphatic glands are often enlarged; but this is due, in my opinion, to septic poisoning, and is not the result of the deposit of leprous material in their substance, though bacilli are frequently found in these glands.

(*c*) Mixed Leprosy.

In this form of leprosy the microbes and their products affect both the skin and the nerves, and the pathology and morbid anatomy are exactly what might be expected from

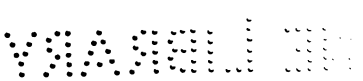
a combination of the two primary forms of the disease. In some cases the tubercular signs predominate; in others the anæsthetic symptoms are most marked. Between the two extremes there are many gradations.

I do not think that mixed leprosy is due to one form being superimposed upon another after the disease has once started, but it is mixed from the beginning. I do think, however, that many cases are wrongly diagnosed at first, and if the symptoms of the one form are very strongly marked, they may almost completely mask the symptoms of the other, so that a mistake can be very readily made in the diagnosis of this form of leprosy. I am of opinion, that in this way mixed leprosy is often mistaken for anæsthetic leprosy.

In some cases the anæsthetic symptoms are well marked from the beginning, but the tubercles are so ill-defined as to be readily overlooked until they grow to a large size and become very perceptible; in such a case the patient would probably be entered in the books as an anæsthetic case, with a subsequent entry stating that tubercles have developed since his admission. I think the tubercles of the syphilitic form of leprosy are often mistaken for the tubercles of tubercular leprosy, as the tubercles of syphilis are not unlike those of some of the mixed cases.

(*d*) Syphilitic Leprosy.

In this form of the disease there is a terrible complication. A patient, with all the pathological signs of syphilis, with its extensive ulcerations of the skin, mucous membranes and bones, and with its infiltrations of the various structures of the body with syphilitic material, is affected



with leprosy in some form. We may have tubercular syphilitic, anæsthetic syphilitic, or mixed syphilitic leprosy, according to the form contracted.

In a mixed form the symptoms are naturally the most severe, and a patient suffering from this form of syphilitic leprosy is in a condition which is almost beyond description.

The syphilitic sores are bad enough in themselves, but when they are accompanied by the terrible deformities of leprosy, the condition of the patient is truly dreadful. The face is disfigured almost beyond recognition, and the body a mass of corruption, a burden to the patient himself, and a loathing to those around him (see Plates XXX. and XXXI.).

I am aware that this last form of leprosy has not been generally recognised by the profession, but I think it is in every way worthy of a separate designation, for though it is produced by a combination of two distinct diseases, yet each malady modifies the other in such a marked manner that the distinctive characters of the primary diseases are lost in the combined disease.

I believe it is by having failed to recognise this form of leprosy that the descriptions of the disease given by most writers are so conflicting, varied, and often misleading.

CHAPTER XII.

SYMPTOMS.

Now that we have considered the pathology and morbid anatomy of the different forms of leprosy, we shall be able to appreciate and comprehend the symptoms to which the changes in the tissues give rise.

There has always, I think, been some confusion in the description of leprosy, because the symptoms of the various forms of the disease have been somewhat mixed, so that to any one not well acquainted with the malady, there appears to be considerable diversity of opinion between the different writers on the subject. The disease is really a simple one, and the symptoms may be readily appreciated if only the different forms of the disease are recognised.

Each form of leprosy has its special symptoms, and a distinct clinical history, from which the individual cases but slightly diverge.

I shall, therefore, give a short account of the clinical history of each of the four forms of leprosy, and mention a few of the most important varieties thereof.

(*a*) Tubercular Leprosy.

The first symptoms of this form of leprosy usually manifest themselves after a cold. The patient while heated

has a cold bath, or he has been out in a snow-storm, or subjected to some severe cold, and becomes feverish. He feels weak and languid, and has fugitive pains about his body and limbs. The patient thinks he is suffering from an ordinary catarrh, or from an attack of simple fever, which he probably neglects, or at any rate takes little notice of, for these first symptoms soon pass over and are probably forgotten.

The febrile symptoms are produced by the poison secreted by the bacilli when they first take up their abode in the body.

The poison enters the blood, and in addition to setting up the premonitory fever, acts upon the terminal branches of the cutaneous nerves, causing vasomotor paralysis of the capillaries, and the consequent formation of erythematous patches in the skin.

These patches are like those of ordinary erythema, they may be large or small, confined to one part, or scattered over the whole surface of the body. Their number and extent probably depend upon the amount of the poison secreted.

The cutis that has become erythematous feels thick and is slightly swollen and tense; the patches may not be irritable, but there is always at this time an excessive secretion of sweat from the parts affected.

The colour of the erythematous patch is fugitive—that is, it disappears on pressure; and the spots themselves are evanescent—that is, they disappear for a time and return again, until eventually most of them fade away and leave no trace behind. With the full development of the rash the febrile symptoms abate and usually pass off.

Some of the patches, however, do not disappear, nor

even fade, but become more and more marked and permanent, until they become stationary.

In one or more of these patches which have become permanent, the first tubercles are formed (see Plate I.).

The first visible sign of a tubercle being formed in an erythematous patch, is often the presence of a blister-like body about the size and shape of a split pea. It is not, however, a blister, for when it is pricked or squeezed no fluid escapes, and it is more or less solid; or the tubercle may be felt in the skin as a small nodule, not unlike that of the first stage of the small-pox eruption.

With each fresh deposit of leprous material this nodule becomes larger until a prominence is formed in the skin, which is visible to the eye; with each deposit there is also at first a certain amount of pyrexia. In some cases, however, the system appears to become inured to the effects of the poison, and the tubercles grow without any appreciable rise in temperature.

Tubercles may form on any part of the body; but, as I have before stated, the most frequent sites are the face, ears, and extremities (see Plate IV.).

The first tubercle is generally formed at the nasal end of the eyebrows (see Plate I.), and as the formation of a tubercle is followed by the loss of hair from the affected part, the swelling of the eyebrow with the loss of hair is one of the first certain signs of the disease.

If, therefore, in a patient who has been suffering from the prodromata, which are in themselves not pathognomonic of leprosy, the eyebrows become thickened, and the hair of the part is thinned or lost, a correct diagnosis is assured.

The tubercles become larger, more prominent, and numerous for about four years (see Plate II.), when they

may be said to be fully developed; after this they commence to ulcerate (see Plate IV.), and to invade the mucous membrane of the mouth and air-passages. The mucous membrane of the lips (see Plate IX.), tongue, hard and soft palates, fauces, and upper part of the larynx, become in turn affected. The tubercles which develop on the mucous membranes generally occur in the order just given.

When the throat is affected the breathing gets obstructed, and the voice, which at first is harsh and croaking, soon becomes sibilant and almost inaudible.

The tubercles which form in the mucous membrane of the nose partly obstruct the passages, so that the voice, at an early stage of the disease, assumes a nasal twang.

As the tubercles grow in the air-passages, the breathing becomes more and more difficult, until the patient is threatened with suffocation.

Those which form on the lips soon ulcerate, and thus give rise to exceedingly painful and intractable ulcers, causing the patient perpetual suffering.

The ulcers in the nose do not, in my opinion, affect the bones, and I am very doubtful whether the cartilages are affected in an uncomplicated case of tubercular leprosy, for I am of opinion that when the cartilages are diseased the malady is complicated with syphilis.

In tubercular leprosy the bacilli are almost wholly confined to the skin and mucous membranes, and, as a rule, the other tissues of the body are not invaded by them, though a limited number are occasionally found in the internal organs.

The nasal ulcers secrete a most fetid discharge which, when combined with the smell of those from the mouth,

face, and extremities, emits an odour very characteristic of the disease, and described by some as sweetish; by others, as like that of a dead body.

The lymphatic glands are often enlarged in leprosy, and not infrequently they become inflamed and suppurate; but this is due to septic poisoning, and not to the deposit in the glands of leprous material. The various parts of the body above-mentioned are the seats of ulcers, from which pus is discharged in considerable quantities, and the glands are naturally soon affected, but the disease in them is not, in my opinion, leprotic.

The internal organs of the body are, also, the seat of amyloid degenerations of the tissues. These changes, too, are not leprous, but brought on by the discharges from the ulcers of leprosy, as emissions caused by any other disease would have the same effect, so that the amyloid degenerations are not leprotic.

The symptoms which these secondary changes in the internal organs give rise to are identical with those caused by the degeneration of these organs from any other cause, so that it will not be necessary for me to discuss them here. Leprosy is essentially a local disease, as far as the bacilli are concerned, and the general symptoms or secondary changes which the ulcerating surfaces give rise to can hardly be considered leprotic.

These secondary changes are, however, the chief causes of death in leprosy.

The patient may die from suffocation, consequent upon the narrowing of the air-passages by tubercular deposits; but this can usually be averted by the performance of tracheotomy or laryngotomy, which I have always found beneficial to the patient, for the opening of the windpipe

below the seat of obstruction, not only gives instant and great relief, but prolongs the life of the sufferer very considerably. A large number of the patients die from exhaustion, brought on by the excessive drain on the system from the numerous sores and ulcers.

Kidney affections, too, carry off a number of the patients of both forms of the disease. Diarrhœa and cardiac dropsy also prove fatal, but, as will be noticed, these are all secondary affections. Many patients, debilitated by leprosy, succumb to acute intercurrent affections, such as erysipelas, bronchitis, pneumonia, &c. It will thus be seen that leprosy *per se* is seldom responsible for the death of a patient.

I have purposely refrained from mentioning any of the local signs of leprosy affecting the extremities, as these are modified considerably by the variety of the disease, or by the different forms which tubercular cases assume. There are five varieties of tubercular leprosy:—

(1) In the most common form, which I have already briefly described, the hands and feet are not, as a rule, much affected, they may be puffed and swollen, but there is usually not much ulceration.

The tubercles on the face, when fully developed, are fairly large, firm, and well defined, and when they ulcerate leave well-defined ragged sores, protected, to a considerable degree, by hard scabs (see Plate III.).

(2) In the second variety of the disease, the tubercles are large, smooth, and soft, but still fairly well defined (see Plate VI.). The cutis appears to be filled with fluid, which is the case, in fact; for if one of these tubercles be incised, a considerable quantity of white glairy fluid escapes. The cuticle is also much attenuated over the

tubercle, so that the capillaries may be seen through it as a fine and delicate network in the cutis beneath (see Plate V.).

The cuticle is, in reality, very thin, and ulcerates readily, forming shallow, painful ulcers. The mucous membrane of the lips soon loses its epithelial coat, and the shallow sores thus formed are exceedingly sensitive and most painful and trying to the patient. The hands and feet in this form of tubercular leprosy are generally very much swollen and puffed, and not infrequently the nails are lost; the tissues under and around the nail becoming inflamed like ordinary onychia, until the nail separates. The ulcer thus formed is very painful, and the patient may generally be seen with all the fingers bandaged to protect the tender and sensitive surfaces from injury. A new nail may grow, but if so, it is very weak, and affords little protection to the finger, being soft, fibrous, and brittle.

In this kind there is also apt to be a certain amount of infiltration and induration of the cutis about the lower part of the legs and feet, which parts readily ulcerate, and thus form shallow and painful, but indolent, wounds, sometimes of very considerable extent.

(3) In the third form of tubercular leprosy the tubercles are small, hard, and well defined. On section they appear to be composed almost wholly of fibrous tissue (see Plate VII.). The summit of a tubercle of this kind is anæmic and pale, and the tubercle does not readily ulcerate; and when it does so, the ulcers are small and ragged. There is little, if any, fluid in a tubercle of this contracted form.

The condition of the tubercle is due to an excessive

amount of fibrous tissue being formed in it by repeated attacks of congestion; there is, in fact, a fibroid degeneration, which almost completely cuts off the supply of blood to the part. If this supply is very defective, the centre of the tubercle suffers most, and becomes necrosed, and a small ragged ulcer is thus formed, which is, however, speedily covered with a scab. In an advanced case, the face is very much disfigured by these tubercles, some of which have pale yellowish summits, others ragged ulcers, and others again have these covered with dirty crusts (see Plate VII.). In this form of leprosy, the hands are, as a rule, not much affected, but occasionally they become swollen and puffed, as in the forms already described; but this form of the disease is generally a sign of chronicity.

(4) In the fourth form of tubercular leprosy, the tubercular infiltration produces a somewhat uniform thickening of the parts affected, the face appears to be uniformly swollen and thickened, and the tubercles are not well defined (see Plate VIII.). The hands and feet are generally swollen and puffed, and are liable to early ulceration. There may also be considerable infiltration of the skin of the extremities with tubercular material.

(5) In the fifth form of tubercular leprosy there is an extensive deposit of tuberculous material all over the body, chiefly, however, on the face, arms, and legs, to the elbows and knees, and more rarely over the front of the trunk and down each side of the spine.

This is a comparatively rare form, and is generally the sign of a chronic case. In the Robben Island Asylum, out of about a thousand cases of leprosy, I have only seen three patients suffering from this variety.

The tubercles are hard and tough, and do not readily

ulcerate, those on the face are generally of the third variety, those on the body are flat, and conform somewhat to the folds of the skin (see Plate IX.). The tubercles do not readily take the form of an ulcer, but, the sweat and sebaceous glands being more or less destroyed, the skin becomes hard and dry, and, as the skin cannot consequently perform its functions well, the internal organs soon become diseased. The probable explanation of the chronicity of this form of the disease is that the mucous membranes do not become much, if at all, affected.

The above are the chief varieties of tubercular leprosy, and the cases belonging to each variety have a fairly distinct clinical history; but, as the varieties are produced by the same bacillus, the action of which is modified only by the severity and nature of the local inflammation, it may very readily happen that in a patient there may be tubercles of each variety coexisting, and one class of tubercles may be changed into another by inflammation. The general tendency is for the tubercles to become harder and firmer with age, but, if the varieties above referred to are recognised, the clinical history of leprosy becomes regular and certain.

The fibroid degeneration of the nodules is a most interesting fact, and one which will, I believe, yet lead to the adoption of the only method by which leprosy can be cured—that is by the production of fibroid degeneration by appropriate treatment.

(*b*) Anæsthetic Leprosy.

The symptoms of this form of leprosy are not as varied as those of the tubercular, for there are no varieties of

anæsthetic leprosy, and each case is exactly like another, except in the severity of the disease, which may, indeed, become arrested in every stage of its existence.

The usual prodromata are followed by the erythematous rash, as in tubercular leprosy; but the rash, unlike that of tubercular leprosy, tends towards permanency and enlargement.

The spots are due to the poison entering the general circulation and acting upon the terminal branches of the cutaneous nerves, and not, I imagine, to the presence in the skin of the bacilli themselves; though, as might be supposed, these may, after the cutis has become weakened by their virus, actually invade this tissue. Of this, however, I am not prepared to speak with any authority, as I have been unable to satisfy myself on this point.

The nerves are at first irritated by the poison, and the spots become sympathetic and painful, but the former are soon more or less disorganised when the latter become gradually anæsthetised.

The whole thickness of the skin is affected by the loss of nerve-power, and each of its component parts becomes atrophied, but the most marked visible change in the skin is the absorption of its pigment (see Plates XXIII. and XXIV.).

In some cases this is wholly removed, and the patch becomes white (this is, however, a rare occurrence), and usually only a portion of the pigment is removed, so that the spots in a dark-skinned person are of a more or less mahogany colour, or they pass through all the shades of brown, red, and yellow.

The poison being in the blood, the spots may form on any part of the body, and there is no special part which

they favour more than another, but they are generally roughly symmetrical (see Plate XXIII.).

These patches are at first isolated, but as they have a tendency to spread, they often coalesce, and so may, in an old-standing case, cover a large area. I have noticed that the centre of a spot (see Plate XXIV.) often resumes its natural colour, and the skin its healthy function, whilst the patch is spreading at the circumference; so that, eventually, the entire spot is represented by a ring of light colour surrounding more or less healthy skin. Occasionally this ring is raised as in Plate XXIV., irritable, and not unlike ringworm, but usually it is faint and ill defined (see Plate XXIII.).

In some cases, when the skin becomes partly restored to health, there appears to be a sort of reaction, and that portion which had been deprived of its colour becomes actually darker than the healthy part surrounding it; but this is an exceptional occurrence, and it usually remains of a lighter colour than the healthy skin.

Why the centre of a patch is thus occasionally restored to health, while the circumference spreads and enlarges, is difficult to say; probably it is because the tissues after a time become accustomed to the effects of the poison, and that only the newly attacked tissues at the circumference feel its effects.

These anæsthetic patches must not be confounded with the anæsthesia of the extremities and face, which is generally unaccompanied by discoloration of the skin.

The areas in question generally become permanent, but, in a few instances, they disappear and leave the skin in a healthy condition.

The hairs in the patches do not fall, but become

bleached and atrophied. The sebaceous glands become atrophied, and there is a marked diminution in the amount of their secretion; consequently the surface of the patch becomes dry, hard, and often scaly.

The general atrophy of the skin affects the sweat glands, and there is a great falling-off in the amount of sweat secreted, which acts as a powerful auxiliary in causing diseases in the internal excretory organs.

The muscles are gradually atrophied, and the adipose tissue absorbed, so that the skin becomes thin and the patient emaciated. Thus it will be seen that the loss of nerve-power causes all the tissues of the skin to become atrophied and wasted. Sensation in the anæsthetic areas is not lost to the same extent in each, or in every part of any single patch.

In some cases there is a complete loss of sensation, in others there is only a numbness of the part; this, of course, depends upon the amount of disorganisation in the nerves.

There are some symptoms that are by no means easy of explanation; for instance, a pin may be thrust into an anæsthetic patch without causing the patient any pain, and yet a feather drawn across the same spot may cause very unpleasant irritation, or, again, a part may be sensible to heat, and not to cold, or *vice versâ*.

Why there should be these differences of feeling, &c., in the anæsthetic patches, it is difficult to explain.

We know that there are three kinds of sensation which we experience by means of the skin—viz., those of touch, temperature, and pain,—but the healthy skin, be it ever so delicately acted upon, responds to all of them, and on every part of its surface. Some parts are undoubtedly more sensitive to certain impressions than others, but

every part of the healthy skin is alive in a greater or less degree to each of the three sensations above mentioned. Yet, in the anæsthetic patch of leprosy, we find that a certain part of the skin is deprived of the power of touch, that another cannot discriminate between heat and cold, though it can feel pain, and that, though a third part may have the sense of feeling, injury does not cause pain. This most interesting point requires elucidation.

The anæsthetic patches, though of great interest and importance from a diagnostic point of view, are of little vital importance, for excepting that they somewhat disfigure the body and throw a little more work on the internal organs, they have but a small, if any, effect on the health of the patient.

Other nerves besides those above referred to are, however, affected by the leprotic poison, and it is by their destruction that so much mischief is done.

Those of the extremities and face become diseased and more or less destroyed, and it is by their disorganisation that leprosy has become such a terror in the land, for this wholesale destruction of the messengers of life cannot but be followed by terrible results.

The dread symptoms commence so insidiously that the disease is generally well advanced before its nature is discerned, and the premonitory symptoms being like those of febricula the disease may very readily pass without recognition in its early stages. Even the erythematous patches may not at first awaken suspicion, and it is generally only when the anæsthesia is detected that the true nature of the terrible disease is grasped. I have already discussed the anæsthetic patches, and will now turn to the much more important changes caused by the destruction of the larger nerves of the extremities and face.

It will not be necessary for me to repeat what I said under the heading of pathology, where I gave my views as to how the nerves became diseased, and how their disorganisation caused necrosis of the bones and destruction of the tissues. I shall, therefore, proceed to a description of the most important symptoms produced by these changes in them.

Usually, the first which would draw your attention to the disease is the anæsthesia of the parts—the patient burns his hands and is unconscious of the fact that he has a wound until his attention is drawn to it by others, or his eye observes it; he finds that he cannot grasp an object firmly, or that his feet are heavy and inclined to drag, or he has a feeling of pins and needles in his arms, legs, or in both.

On thinking over the matter or being questioned on the point, he may remember the premonitory symptoms—the fever and the rheumatic pains,—and on examination the patches may be seen. By gauging the sensibility of the extremities a distinct loss of power will be detected.

The little finger is the first generally to become anæsthetic, and also to become contracted (see Plate XII.), but it is soon followed by the ring, middle, and index fingers, the thumb being usually the last to become flexed (see Plate XVII.).

The order in which this contraction occurs is liable to variation, for, according to the patients, the thumb is occasionally the first to become affected, but this statement is apparently not borne out by the appearance of those in the asylum.

The degree of contraction is in direct proportion to the amount of disease in the nerves, it may be only very

slight (see Plate XVII.), or the fingers may be firmly clinched on the hand (see Plate XXIX.). Commencing at the last joint of the finger (see Plate XII.) it is always greatest at the second joint (see Plate XXIX.), and the metacarpo-phalangeal joints are not, as a rule, much affected.

As the joints become flexed they become rigid, so that they cannot be straightened by force; this is not, however, due to contraction of the tendons.

The skin of the extremities, excepting that it is anæsthetic, is not at this stage much interfered with. Being deprived of some of its nerve-power it is, however, liable to injury. The cuticle has a lowered vitality, so that the slightest injury causes a breach of surface; the consequence is, that even at an early stage of the disease the hands and feet are covered with wounds and scars, but these injuries are not leprotic.

The boot chafes the heel and causes a blister—a long walk, which, when the patient was in good health would have no evil effect, now produces blisters and ulceration of the feet; the hands are burnt by fire, hot water, hot utensils, or even by a tobacco pipe, and wounds are formed, but all these are not leprous, for they heal readily: they are due to a defective nerve-supply and only secondary to the disease.

I am of opinion that the bullæ, of which so much has been written, are the results of accidents, and are not due directly to the effects of the poison secreted by the bacilli; they are accidental to the disease and are produced by injury of some kind.

Heat, which in an ordinary healthy skin would produce no effect or only perhaps a slight redness of the part,

would in one deprived of its nerve-power probably produce extensive ulceration of the tissues, so that the application of heat only a little higher than that of the body would produce a blister in leprosy, in the same way as pressure would readily cause destruction of the tissues.

We know how even the temporary lowering of the vitality of a part will cause extensive sloughing of the tissues, as, for example, in acute fevers; how much greater, then, must be the effect when the tissues are permanently deprived of this power? It is for these reasons that the hands and feet of lepers are so often covered with sores and wounds, even at an early stage of the disease.

The bullæ may form on any part of the body, but are chiefly confined to the exposed parts. They are liable to occur at all stages of the disease, but are more numerous in its early career, probably because the patient is at that time more exposed to injury. The wounds which the bullæ cause are never very deep and heal readily, and the scar produced by them is depressed and of a light colour, which is probably due to the fact that the repair of the injured part is not very complete (see Plate XVIII.).

In addition to the contraction of the parts and anæsthesia of the skin the tissues become atrophied, the adipose tissue absorbed, and the muscles much reduced in size, so that the bones become prominent and well defined, the skin seeming to be tightly drawn over them (see Plate XXIX.). The appearance of the part affected is like that of extreme emaciation. The most prominent muscles of the healthy hand are those forming the ball of the thumb and the abductor minimi digiti; when these become atrophied the hand assumes a peculiar square shape (see Plate XXIX.), and when this is combined

with atrophy and contraction of those of the fingers the hand assumes a shape very characteristic of leprosy, called the "main-en-griffe."

All the muscles of the hand are affected, but, as a rule, the extensors have to give way to the flexors, so that there is a uniform contraction of the joints; but the fingers are also, more or less, twisted on their own axis (see Plate XIV.), and drawn across each other according to the degree to which the special muscles are affected, so that even if there is no destruction of the bones and no ulceration of the skin or loss of bone there may be very great deformity of the hands and feet. The phalanges are the first to become diseased, and of these the first is the second phalanx. The bone becomes diseased and sets up inflammation in the tissues surrounding it, the finger becomes swollen (see Plate XV.), tense, and glistening, the swelling soon shows signs of suppuration, and the abscess, if left to itself, soon bursts, discharging a considerable amount of dark, glairy pus, leaving a wound (see Plate XIII.) which leads down to the diseased bone.

As soon as the dead bone is removed by operation or otherwise the wound heals. One bone after another is thus attacked and destroyed, and the patient is seldom free from suppurating wounds until a point is reached where the parts are supplied with sufficient nerve-power to keep them alive if not healthy, and at this point ulceration ceases (see Plate XVIII.).

Occasionally the inflammation is so acute about a bone that the finger becomes gangrenous and is lost, but usually it is only the bone that is lost, and the flesh contracts until, in many patients, the nail rests upon the metacarpo-phalangeal joint. This, I think, points to the

fact that it is the death of the bones that causes destruction of the tissues.

I cannot agree with those who state that the ulceration starts on the surface and extends to the bone.

There are two kinds of ulcers in anæsthetic leprosy—those due to injury to the skin, and those due to necrosis of the bone, which is the result of the disease in the nerves. A burn may be so severe as to cause destruction of the tissues down to the bone, and even of the bone itself, but such wounds cannot be considered leprotic, they are accidental to the disease. Leaving, therefore, such out of consideration, I am of opinion that all other ulcers on the extremities of anæsthetic patients have their origin in the bones.

The bone becomes necrosed and sets up inflammation, and a sinus is formed to give exit to the products of this inflammation.

If the bone thus necrosed is a phalanx it is readily detached from its fellows; but if it is a metacarpal bone, for instance, it is by no means so readily got rid of, and the sinus remains open for years; and it stands to reason that the more firmly the diseased bone is impacted, the more chronic will be the wound or sinus.

These chronic sinuses are called perforating ulcers, and a very expressive name it is, for they do perforate the flesh to give exit to the dead bone.

I think too much stress has been laid upon these perforating ulcers, and their importance much magnified. Perforating ulcers are certainly found in almost all cases of anæsthetic leprosy, but this is only because in this form of leprosy the nerves are destroyed, but they are not pathognomonic of it, for any disease which is accompanied

by destruction of the nerves is also accompanied by perforating ulcers. I believe that in many cases, also, the perforating ulcers exist when leprosy has long ceased to, and that, therefore, the presence of a perforating ulcer in a patient who has suffered from anæsthetic leprosy is no proof that the disease is still there.

When the disease starts in the nerves there is often, in fact almost invariably, at first hyperæsthesia of the parts affected, and the patient is tormented by neuralgic pains and extreme sensibility of the affected limbs, the slightest jar or knock will send a shock like that from an electric battery through the body. These pains are often very severe and of long duration, so that the patient is often worn out by them, as they prevent sleep.

The stage of hyperæsthesia is followed by anæsthesia and ulceration.

The most frequent seats of this neuralgic pain is about the eyes and on the cheeks.

Some of the observers who have studied leprosy mention that they have seen cases in which the mucous membrane of the mouth and throat has become anæsthetic; but I have not met with this form of paralysis, for I have in anæsthetic leprosy invariably found the mucous membranes healthy.

The paralysis of the nerves of the face commences at a comparatively early stage of the disease, and the resulting deformities are very characteristic of leprosy.

The facial branches of the seventh nerve and the first division of the fifth are those usually affected.

The paralysis of the infraorbital branch of the seventh nerve causes atrophy of the lower eyelid and paralysis of the orbicularis (see Plates XXII. and XXVIII.).

The patient is unable to close the eyes, and in his attempts to do so he contorts the face very considerably, and the eyeball rolls upwards whilst the eyelid remains motionless.

If the fifth nerve is also affected the lachrymal gland becomes atrophied, and the secretion of tears is stopped; but usually this nerve is not diseased, and the tears, diverted from their natural course by the eversion of the eyelid, flow down and excoriate the cheek.

The eye itself being insufficiently protected becomes inflamed, the cornea soon becomes opaque (see Plate XX.), and not infrequently the eye is lost through ulceration of the cornea; but in most patients, though much inflamed and disfigured, it is not destroyed.

One of the first signs of paralysis of the orbicularis is that the eye stares peculiarly, it is a glassy fixed look very characteristic of this stage of anæsthetic leprosy (see Plate XXI.).

The buccal branches of the seventh nerve become affected, and the cheeks and lips flaccid and pendulous, which naturally gives the patient a most doleful expression of countenance, very well seen in Plates XVI., XX., XXII., and XXVIII.

The staring eyes, averted eyelids, expressionless cheeks, and flaccid lips give the anæsthetic leper an appearance which, when once seen, can never be forgotten.

In a severe case the lips puff out while the patient is speaking, as in ordinary facial paralysis, and I have seen one patient (see Plate XX.) in which the paralysis was so complete that he was unable to articulate without raising the lower jaw with the stumps of his hands.

The paralysis of the lips often causes the teeth to be

exposed in a most unpleasant manner (see Plate XXX.). That of the legs and feet causes the patient to walk in a peculiar manner, and with a gait very characteristic of the disease.

The extensors of the toes being also the flexors of the ankle-joint, and as the extensors are the first to become affected in anæsthetic leprosy, it follows that the patient is unable to flex the ankle-joint or raise the toes from the ground. In his efforts to do so the patient raises the whole foot from the ground by bending the knee and bringing it forward; when this has been accomplished and the toes are clear of the ground he at once swings the leg forward and flops the foot down, the toes first coming in contact with the earth.

The toes are often all removed by ulceration, and in many cases the foot itself is gone; but in most of the cases the disease does not go farther than to cause the loss of one or more of the toes, and the formation of a perforating ulcer on the sole of the foot.

In one case all the bones of the feet, except a portion of the os calcis, were removed, as also the lower third of both tibia and fibula; in this case, however, not by ulceration, but by absorption. I have produced the portrait of this patient in order to show a similar condition in the upper extremities (see Plate XX.). The paralysis is often so complete that the hands and feet cannot be moved except by being flung about at the end of the stump like a flail.

I have photographed a number of patients to show the various deformities which I have attempted to depict in these pages; with each photograph which I have produced I have given a short history of the patient and of the case,

and I have in each instance pointed out the special features which the photograph is meant to illustrate.

During the course of leprosy the patient is subject to all manner of intercurrent affections, which it is needless for me to describe in these pages, which are written solely to assist those who are not practically acquainted with leprosy in forming a correct idea as to its nature and appearance.

The lowered vitality of so much of the body causes the leper patients to feel the cold very much, and if they can they will be constantly crouching round a fire.

As a rule, they are also subject to various forms of dyspepsia, and especially with pyrosis.

I have not found the generative organs especially affected in anæsthetic leprosy.

All operations are well borne, and wounds thus produced heal very readily, usually by first intention.

In chronic cases the patches may disappear and leave no trace behind, but usually the skin of even those patients in whom the disease has been arrested presents a faintly mottled appearance.

(*c*) Mixed Leprosy.

It is hardly necessary for me to describe the symptoms of this form of the malady, as they are exactly the symptoms of anæsthetic leprosy, added to those of the tubercular form of the disease; but, as I have already stated, there are innumerable variations in the way in which these two primary forms can join, and it is very seldom that anæsthetic and tubercular are combined in the same degree or proportion in a patient.

Usually one form or the other predominates.

In some cases the symptoms of one class are almost entirely masked by those of the other, and in this way, unless the greatest care be taken, an incorrect diagnosis is made in the first instance, and it is only when the disease is far advanced that the error is detected; but the mistake is not practically grave, as the mixed form is, like the tubercular form, a severe one. It is a strange thing, however, that when the two forms are combined the disease appears to assume a more chronic type than when the tubercular form exists alone. One would have fancied that if anæsthetic leprosy were added to tubercular leprosy the patient would stand a worse chance; but this is not the case, the presence of anæsthetic leprosy seems in some way to retard the growth of the bacilli in the tubercles.

The symptoms in the mixed form are, however, very severe, for in addition to the ulcers and deformities of anæsthetic leprosy, we have the tubercles and infiltrations of the tubercular form (see Plates XXVI. and XXVIII. for two typical cases of mixed leprosy, one in which the tubercular symptoms predominate, the other in which the anæsthetic symptoms are the most marked).

(*d*) Syphilitic Leprosy.

The poison of syphilis acts upon the skin in very much the same manner as does the poison of leprosy, and it produces tubercles and a rash not unlike it.

The syphilitic tubercles, however, ulcerate at an early stage of the disease, and the face becomes covered with ugly shallow ulcers which, when they heal, leave the skin

much deformed. When this is complicated by leprosy a very ragged face is the result. Such a face is well represented in Plate XXXI.

In these syphilitic leprosy cases the mouth and throat become much affected, the hair is removed from the scalp in large patches, the bones in various parts of the body become necrosed, the lymphatic glands are enlarged and often suppurate, indolent abscesses are formed in various parts of the body, the bones of the nose are soon lost, and the nose itself is removed by ulceration, these symptoms being due almost entirely to the syphilitic poison. Add to these disfigurements the deformities of any form of leprosy, and symptoms are produced almost too terrible to behold. The head bald in patches; the eyes shrunken, and in most cases disorganised, and almost hidden by a swollen face; the face swollen, scarred, puckered, and covered with ragged wounds and scabs; the mouth, with retracted lips and prominent teeth; foul discharges pouring from the mouth and nose; abscesses about the neck and body; hands reduced to mere suppurating stumps; and feet a shapeless mass of corruption, and you have syphilitic leprosy in its last stage (see Plate XXXIV.).

I have, by a selection of photographs, endeavoured to illustrate the symptoms and signs of the various forms of syphilitic leprosy. The feet in Plate XXXIV. will give the reader an idea of the deformities caused by this form of leprosy.

CHAPTER XIII.

CAUSE OF DEATH.

As will be seen from the following list, leprosy *per se* is responsible for very few deaths, most of the patients dying from secondary diseases. Of 107 patients who died in the Robben Island Asylum since 1892 the following were the causes of death :—

Accident	1	Marasmus	12
Apoplexy	1	Nephritis	7
Bronchitis	9	Peritonitis	2
Diarrhœa	10	Phthisis	12
Dropsy	2	Pleurisy	6
Dysentery	6	Pyæmia	4
Enteritis	1	Pyrexia	1
Erysipelas	17	Spasm of Glottis	1
Gastritis	1	Syncope	2
Hæmorrhage	1	Ulceration of Throat	1
Laryngeal Affections	10		

It will be seen that erysipelas was responsible for the largest number of deaths. Marasmus, due to exhaustion from excessive discharges, and phthisis come next.

The phthisis was due to the tubercle bacillus, and not to that of leprosy.

CHAPTER XIV.

DURATION.

THE following tables have been prepared to show the duration of the disease in those patients who were admitted into the Robben Island Asylum since 1871.

Unfortunately, in the old records of the asylum, no distinction was made in the case books between the various kinds of the disease, so that the figures only point to the duration of leprosy without any reference to the form of it.

From 1871 until the 30th of June, 1894, excluding those patients who were again discharged from the asylum, there were admitted into the Robben Island Leper Settlement 964 patients, of whom 402 died.

Of the 402 patients who died

151	died	within	1	year	of	admission.
140	„	„	2	years	of	admission.
33	„	„	3	„	„	„
25	„	„	4	„	„	„
16	„	„	5	„	„	„
12	„	„	6	„	„	„
6	„	„	7	„	„	„
10	„	„	8	„	„	„
5	„	„	9	„	„	„
1	„	„	10	„	„	„
1	„	„	11	„	„	„
1	„	„	12	„	„	„
1	„	„	16	„	„	„

The average duration of the residence of these 402 patients in the hospital was, therefore, nearly two years and six months.

It will be seen that of the patients who died in the hospital during the last twenty-four years two only resided in the asylum longer than eleven years.

Of the 562 patients who were in the asylum on the 30th of June, 1894,

44	had been in the	Asylum	less	than	1	year.
196	„	„	„	„	2	years.
248	„	„	„	„	3	„
19	„	„	„	„	4	„
6	„	„	„	„	5	„
21	„	„	„	„	6	„
4	„	„	„	„	7	„
8	„	„	„	„	8	„
5	„	„	„	„	9	„
1	„	„	„	„	10	„
3	„	„	„	„	11	„
7	„	„	„	over	11	„

So that of the 562 patients only seven had been in the asylum longer than eleven years.

Several of these cases are of the self-cured class, and will probably live to an old age in the asylum, unless they are carried off by some intercurrent affection.

These two tables indicate that a large number of the leper patients die within two years of admission.

I have prepared the following table to show since 1891 how long the patients who were admitted resided in the asylum after admission and until they died:—

	1892.	1893.	1894.
Died within 1 year	23 ..	65 ..	31
„ „ 2 years	3 ..	26 ..	36
„ „ 3 „	6 ..	7 ..	25

	1892.	1893.	1894.
Died within 4 years	2	5	1
„ „ 5 „	1	3	2
„ „ 6 „	3	2	—
„ „ 7 „	—	2	—
„ „ 8 „	1	2	—
„ „ 9 „	1	1	—
„ „ 10 „	—	1	—
„ „ 13 „	—	—	1

The average stay for 1892 was 2 years and 4½ months.
„ „ „ „ 1893 „ 2 „
„ „ „ „ 1894 „ 2 „ „ 2 „

These figures would indicate that leprosy is a much more acute disease, and more rapid in its course than one would suppose from a perusal of the literature on the subject, and I am of opinion that its duration as generally given is too high, and that the average duration is incorrectly arrived at by including in the calculation a number of cases in which the disease is permanently arrested.

In all asylums there are undoubtedly many of these cured cases, and if only a few of them are included in the calculation, the average duration obtained would naturally be much higher than it should be.

There are patients in the asylum under my charge who contracted the disease upwards of sixty years ago, and who, though they are in my opinion self-cured, and have been so for many years, are still classed as lepers, for medical men, as a rule, do not acknowledge that they are cured, for the old saying, "Once a leper always a leper," has been too long impressed on their minds.

I have prepared the following tables to show the duration of the disease in cases in which I have been able to obtain fairly reliable information.

The tables show when the disease was contracted, when

the patient died, the duration of the disease, the date of admission into the asylum, and the immediate cause of death in each case.

TUBERCULAR LEPROSY.

Initial.	Contracted. Year.	Died. Year.	Duration. Years.	Admitted. Year.	Disease.
D.	1891	1894	3	1893	Erysipelas.
U.	1890	1893	3	1893	Pyrexia.
J. C.	1887	1893	6	1893	Cancer.
J. F.	1888	1894	7	1893	Erysipelas.
P.	1890	1893	3	1892	Marasmus.
C. M.	1890	1893	3	1892	Marasmus.
W. R.	1884	1893	9	1892	Erysipelas.
P. B.	1887	1895	8	1892	Throat Affection.
S.	1891	1894	3	1892	Erysipelas.
J. H.	1885	1893	8	1892	Throat Affection.
S. F.	1889	1893	4	1892	Erysipelas.
J. F.	1884	1894	10	1892	Apoplexy.
N.	1887	1893	6	1892	Enteritis.
K. K.	1884	1893	9	1893	Dyspnœa.
K.	1892	1894	1½	1892	Dropsy.
J. P.	1889	1893	4	1892	Erysipelas.
B. S.	1889	1894	5	1892	Pleurisy.
K. P.	1888	1893	5	1892	Marasmus.
O. S.	1884	1893	9	1892	Marasmus.
S. K.	1887	1894	7	1893	Marasmus.
K. S.	1882	1895	13	1893	Nephritis.
O.	1885	1894	9	1892	Erysipelas.
W. R.	1888	1893	5	1890	Marasmus.
M.	1883	1893	10	1893	Peritonitis.
C.	1885	1894	9	1891	Bronchitis.
J. S.	1889	1894	5	1891	Dysentery.
D. P.	1891	1893	2	1891	Nephritis.
S.	1891	1895	4	1892	Nephritis.
A. K.	1891	1894	3	1892	Erysipelas.
G. J.	1890	1895	5	1892	Pyæmia.
J. G.	1891	1893	2	1892	Dyspnœa.
J. S.	1889	1895	6	1893	Pyæmia.

Initial.	Contracted. Year.	Died. Year.	Duration. Years.	Admitted. Year.	Disease.
A. W.	1890	1894	4	1893	Dysentery.
M.	1892	1894	2	1894	Dyspnœa.
S.	1890	1895	5	1893	Dyspnœa.
J. J.	1894	1895	1	1894	Dyspnœa.
Z.	1889	1894	5	1893	Spasm of Glottis.
A. P.	1890	1894	4	1893	Bronchitis.
P.	1882	1894	12	1893	Phthisis.
J.	1886	1894	8	1893	Dysentery.
K.	1890	1895	5	1894	Peritonitis.
Z.	1892	1895	3	1894	Nephritis.
W.	1893	1894	1	1894	Diarrhœa.
J.	1891	1895	4	1894	Dysentery.

Of these forty-four patients who suffered from the tubercular form of the disease, the average duration of the disease was 5·5 years nearly.

ANÆSTHETIC LEPROSY.

Initial.	Contracted. Year.	Died. Year.	Duration. Years.	Admitted. Year.	Disease.
S. M.	1874	1893	14	1893	Diarrhœa.
J. O.	1870	1895	25	1893	Phthisis.
B.	1878	1895	17	1892	Phthisis.
P. B.	1878	1893	15	1892	Nephritis.
D.	1877	1894	17	1892	Erysipelas.
P.	1885	1893	8	1892	Marasmus.
W.	1886	1894	8	1892	Accident.
J. S.	1887	1894	7	1893	Dysentery.
B.	1873	1894	21	1892	Dyspnœa.
R. P.	1874	1893	19	1893	Phthisis.
H. J.	1880	1894	14	1893	Phthisis.
C.	1883	1893	10	1893	Diarrhœa.
J.	1890	1895	5	1893	Marasmus.
G. P.	1864	1895	31	1894	Marasmus.
M.	1889	1894	5	1894	Nephritis.
V.	1887	1895	8	1893	Dropsy.

Initial.	Contracted. Year.	Died. Year.	Duration. Years.	Admitted. Year.	Disease.
Z.	1892	1894	2	1893	Bronchitis.
K. K.	1885	1893	8	1890	Nephritis.
P. A.	1885	1895	10	1889	Phthisis.
H. K.	1873	1895	22	1889	Diarrhœa.
E.	1867	1894	27	1890	Diarrhœa.
S. M.	1885	1893	8	1892	Heart Disease.
J.	1885	1894	9	1892	Diarrhœa.
W. B.	1885	1894	9	1893	Erysipelas.
A.	1891	1893	2	1891	Marasmus.
J. G.	1891	1893	2	1891	Pyæmia.
D. V.	1884	1894	10	1892	Phthisis.
S.	1887	1893	6	1892	Phthisis.
J. S.	1887	1894	7	1892	Phthisis.
W.	1879	1894	15	1892	Pyæmia.
J. C.	1880	1893	13	1885	Bronchitis
A. F.	1880	1893	13	1882	Pyæmia.
W. M.	1884	1893	9	1887	Pyæmia.
J. J.	1885	1893	8	1892	Pyæmia.
S.	1883	1893	10	1890	Pleurisy.
A. E.	1878	1894	16	1893	Diarrhœa.
P.	1882	1893	11	1892	Bronchitis.
T. M.	1888	1893	4	1893	Bronchitis.
J. H.	1884	1893	9	1886	Syncope.
F. R.	1884	1893	9	1892	Nephritis.
K. J.	1890	1894	4	1893	Dysentery.

Of these forty-one patients who died suffering from anæsthetic leprosy, the average duration of the disease was eleven years and five months nearly. Many of these patients were, however, of the chronic class, and as time goes on, and all the acute cases die off in the hospital, as they have been doing during the last three years, the average duration of the stay of those patients who remain in the hospital will be much increased.

MIXED LEPROSY.

Initial.	Contracted. Year.	Died. Year.	Duration. Years.	Admitted. Year.	Disease.
G. S.	1888	1893	5	1893	Nephritis.
N. S.	1886	1893	7	1892	Marasmus.
J. V.	1877	1893	16	1892	Diarrhœa.
E. T.	1884	1893	9	1888	Diarrhœa.
C. S.	1886	1894	8	1893	Erysipelas.
B.	1890	1893	3	1893	Bronchitis.
J. R.	1886	1893	7	1893	Dyspnœa.
J. J.	1870	1893	23	1892	Heart Disease.
D. N.	1877	1893	16	1892	Spasm of Glottis.
W. M.	1885	1894	9	1892	Phthisis.
L. L.	1880	1892	13	1888	Diarrhœa.
J. T.	1882	1893	11	1886	Diarrhœa.
B. G.	1883	1894	11	1893	Phthisis.
P. J.	1877	1893	16	1886	Diarrhœa.
J. W.	1887	1894	7	1892	Erysipelas.
J. A.	1890	1893	3	1891	Erysipelas.
F. R.	1885	1895	10	1891	Pleurisy.
T. N.	1883	1893	10	1892	Bronchitis.
C. D.	1887	1894	7	1892	Pleurisy.
B.	1889	1893	4	1892	Heart Disease.
J. K.	1882	1893	11	1892	Erysipelas.
A. T.	1888	1893	5	1893	Diarrhœa.
M. P.	1893	1894	1	1893	Phthisis.

The average duration of the disease in these twenty-three cases of mixed leprosy was nine years and three months nearly.

From these figures it will be seen that the tubercular form of the disease is the most rapid in its course, and that very few patients live longer than eight years after contracting it. In the mixed form the severity of the disease and its duration depend a good deal upon the complication; when the tubercular form predominates the disease is naturally of a more acute type than when the

anæsthetic form is in the ascendant. As syphilitic leprosy has not been hitherto recognised as a distinct form of the disease, many of the syphilitic cases have been classed among the mixed cases. It is worthy of note that in the syphilitic form of leprosy the disease tends to assume a chronic form, which may be due to the presence of two bacilli in the system.

The last three tables are interesting in that they show the immediate cause of death in the various forms of the disease. In the tubercular form erysipelas figures very largely as a cause of death; in the anæsthetic form diarrhœa, phthisis, and pyæmia; and in the mixed form there is a mixture of these causes.

CHAPTER XV.

PROGNOSIS.

LEPROSY is due to the presence in the system of a bacillus, upon which, hitherto, nothing that has been tried has had much, if any, effect, so that the disease is a very fatal one, and the prognosis, therefore, bad. Leprosy commences very insidiously, and progresses steadily from bad to worse, and on to a fatal termination. In the tubercular form death usually takes place within eight years; and in the anæsthetic form, within eleven years. A few tubercular cases have exceeded this limit, and the disease has been unduly prolonged by the effects of the intercurrent affections. In the anæsthetic form about 5 per cent. of the cases become permanently arrested.

As will be seen from the table on p. 86, death occurs at all seasons of the year with inexorable regularity, for nothing stays the course of the disease.

The self-cured cases of anæsthetic leprosy do not die of leprosy, or even its usual complications.

Table showing the number of deaths during each month since 1871, also the total deaths during the respective months and years.

Year.	Months. 1	2	3	4	5	6	7	8	9	10	11	12	
1871 ..	1	1	1	0	1	0	1	3	0	0	0	1	= 9
1872 ..	1	1	1	1	1	2	0	1	0	1	0	0	= 9
1873 ..	0	0	0	0	1	2	1	1	1	1	0	0	= 7
1874 ..	1	1	1	2	2	0	1	3	1	1	1	0	= 14
1875 ..	2	0	3	2	0	0	0	0	0	1	0	0	= 8
1876 ..	1	0	1	0	1	1	0	0	1	0	0	0	= 5
1877 ..	0	1	1	1	0	1	1	0	0	1	0	2	= 8
1878 ..	1	2	0	1	0	0	0	0	1	1	1	0	= 7
1879 ..	3	0	0	2	1	0	1	0	0	0	0	0	= 7
1880 ..	1	1	1	1	0	0	0	0	1	1	1	3	= 10
1881 ..	0	0	1	0	1	2	0	2	1	2	3	0	= 12
1882 ..	0	0	3	1	1	0	5	1	2	0	2	2	= 17
1883 ..	1	1	0	0	1	1	0	1	0	1	0	0	= 6
1884 ..	1	0	0	0	1	0	0	3	0	3	0	2	= 10
1885 ..	3	1	1	2	1	0	1	0	1	1	0	2	= 13
1886 ..	0	0	0	0	1	1	3	1	1	0	2	0	= 9
1887 ..	2	2	0	1	0	1	1	1	2	1	1	0	= 12
1888 ..	1	1	1	0	1	0	4	3	0	0	3	0	= 14
1889 ..	2	2	3	1	0	2	2	2	3	6	5	3	= 31
1890 ..	4	0	3	1	1	2	2	1	0	1	4	4	= 23
1891 ..	2	1	3	0	3	0	3	2	3	2	1	1	= 21
1892 ..	2	5	4	4	1	4	1	5	4	3	3	4	— 40
1893 ..	11	8	5	5	9	10	5	5	14	14	14	14	= 114
1894 ..	7	4	4	4	5	13	12	8	7	13	5	14	= 96
Total	47	32	37	29	33	42	44	43	43	54	46	52	= 502

CHAPTER XVI.

DIAGNOSIS.

THE changes produced by the bacilli in an advanced case of any form of leprosy are so marked that it is almost impossible for any one who has seen a case, or even the photograph of one, to make a mistake; but in a mild case, and in the earlier stages of the disease, it is by no means easy sometimes to make a correct diagnosis.

I think it advisable to discuss the diseases which can be mistaken for the different forms of leprosy separately. The initial pyrexial symptoms with which the disease is generally ushered in, may be mistaken for febricula, or some form of low fever. During this stage it would be almost impossible to make a correct diagnosis.

If the fever is followed by an erythematous rash, some of the patches of which tend to become fixed, suspicion of the true nature of the disease should be aroused in the mind of a careful observer, especially in a country in which the disease is prevalent; but even then a definite opinion could not be pronounced until the tubercles commence to form, or the parts become anæsthetic. The first diagnostic symptom of tubercular leprosy being the thickening of the skin, with loss of hair, and in anæs-

thetic leprosy, paralysis of sensation, and, perhaps, motion.

Gutta rosacea, so common amongst females, especially of the farming community, may be readily mistaken, in its advanced stages, for the erythema of the first stage of leprosy; but this eruption is generally on the chin, cheeks, and nose, and the eyebrows are seldom affected. The history of rosacea is also unlike that of leprosy. It commences as a vivid flush, which, as it becomes chronic, assumes a darker hue, and is accompanied by a sensation of burning, throbbing pain, not frequently present in leprosy. It is also a disease of middle life, and is usually accompanied by some general ailment or disorder of the economy.

I think the chief diagnostic differences between the two eruptions is that, in rosacea there is a more uniform thickening of the skin over a larger area; the colour of the eruption is also more uniform than that of the erythema of leprosy; the hairs are not lost, and scales are apt to form on the patch. In leprosy the patch is darker in the centre, and from this dark point the colour fades into the surrounding skin, and the hairs are soon lost from the tuberculous patch.

It is sometimes necessary to give an opinion and make a correct diagnosis between these two diseases under very trying circumstances. For instance, when the member of a leprous family is the subject of an eruption, which it is by no means an easy matter to distinguish from the erythema of leprosy, the anxiety of an indefinite opinion, under such circumstances, is naturally very great, and for these reasons I have thought it necessary to discuss the points of difference, and also of resemblance,

of these two affections at some length, especially as I know of no other disease which can be so readily mistaken for leprosy as rosacea.

Kelis has been mistaken for tubercular leprosy, but its resemblance to a cicatrix, its size and elevation, with the peculiar soft feeling it presents on the surface, and the hard, fibrous base, with the absence of all signs of congestion, are sufficient to distinguish it from leprosy. The swelling in kelis is also local, and may be in the site of an old wound; and it is not bilateral as are the tubercles of leprosy (see Plate XXXV.).

General thickening of the skin, due to chronic inflammation, has been mistaken for leprosy, but in this case the thickening is too general and too uniform to be mistaken for leprosy by anyone who has any knowledge of the latter disease; besides, in chronic dermatitis, there is an absence of tubercles.

Elephantiasis Arabum may be mistaken for tubercular leprosy, but in this disease the thickening of the skin is greater than in leprosy, and the swelling is generally confined to the lower extremities and scrotum (see Plate XI.).

Many diseases have been mistaken for anæsthetic leprosy by even the most careful observers, and it is undoubtedly difficult in some cases to pronounce a definite diagnosis, or opinion. In one patient who was sent to the asylum as a leper the nerves of both forearms were disorganised, the parts were anæsthetic, and the patient had the characteristic *main en griffe*, and he had, in addition, paralysis of both legs. The case was, undoubtedly, very much like anæsthetic leprosy, but the nerves were not thickened, the eyes were not affected, and there were no anæsthetic patches nor absorption of

bone. The nerves had not been destroyed by the leprotic poison.

It is difficult to distinguish between ulcerations and the perforating ulcers due to the ordinary destruction of the nerves, and those due to the destruction of the nerves by the leprotic poison, but in these cases some of the characteristic symptoms of leprosy are always wanting.

Any nerve may be injured and lead to ulceration, but the lesions would probably be local and unilateral, and there would be no discoloured anæsthetic patches.

Paralysis agitans has been mistaken for leprosy. One patient in the leper wards had this form of paralysis. She was unable to walk or even leave her chair. Constant friction had caused extensive ulceration of one foot. Both feet were very much deformed, and the hands contracted, but she had no paralysis of sensation, no anæsthetic patches, and her eyes were unaffected.

The deformities, due to rheumatic arthritis, have been mistaken for those due to leprosy, but the absence of anæsthesia should make the diagnosis easy.

The ulcers and deformities, due to burns and other injuries, have led to a wrong diagnosis; but no medical man, with ordinary care, should mistake the signs.

One patient was sent to the island because he had lost one of his great toes by necrosis; another because he had lost his feet by gangrene. The latter patient was kept in the Leper Asylum for many years without any suspicion having apparently been raised as to the true nature of the disease or deformity.

Tertiary syphilis is not infrequently mistaken for leprosy, and it is not always easy to distinguish between a severe form of syphilis and anæsthetic leprosy. In the

former there is not much anæsthesia, and the discoloured patches are not anæsthetic as they always are in leprosy, and the hands of a syphilitic patient, though often much disfigured, are not paralysed.

Lupus exedens has been mistaken for leprosy, but with a moderate amount of care the ulcers should not be mistaken.

CHAPTER XVII.

TREATMENT.

LEPROSY is due to the bacillus lepræ; therefore, in order to cure the disease, this bacillus must either be destroyed or rendered inoperative in some way.

No remedies that have hitherto been tried have brought about either of these much-to-be-desired results, and this, not because the remedies are not good, but because they cannot be effectually applied.

The bacilli multiply in the body, and secrete a poison which circulates through the system, and nothing that has been done can either dislodge them or modify the effects of their poison.

Innumerable drugs have been tried for the cure of leprosy, and many specifics vaunted, but each has in its turn been cast aside as useless.

We do not want new germicides, for the Pharmacopœia is replete with powerful disinfectants and antiseptic drugs, but we do want a method by which the known drugs can be applied, for, if we could only get at the microbes, we could destroy them.

If we cannot cure the patients, however, we can do a great deal to ameliorate their condition by appropriate

treatment. Leper patients should in all cases be accommodated in sunny, well-ventilated rooms, and their surroundings should be in the best sanitary condition.

Hospitals for leper patients should be erected in warm, dry localities, as many of the patients suffer intensely from the cold, and a large number of them develop chest complaints. The patients should be warmly clad, and should invariably wear flannel or woollen underclothing, as they are not only very susceptible to the vicissitudes of the weather, but I believe colds and draughts favour the growth of the bacilli by disturbing the cutaneous circulation, therefore they should be so clad as to insure, as far as possible, an equable temperature to the skin.

The wards should for the same reason be large and airy, with plenty of cubic space per patient, so as to avoid through currents of air and draughts. They should also be well provided with heating appliances, open fires are not, however, advisable, as the patients are liable to burn themselves very severely on account of the anæsthesia of the extremities.

The patients should be given a generous diet, in which butter and other oleaginous matter should be well represented; good bread, fresh meat, fish, milk, eggs, and vegetables are indispensable.

Patients almost invariably improve when first admitted into a good asylum, which is due to good living and healthy surroundings, but the improvement thus brought about is only temporary, for do what we can the disease generally goes on from bad to worse, until the patient is reduced to a mere wreck, or the disease becomes arrested.

I have noticed that a mask applied to a tuberculous

spot causes a certain amount of local improvement, the tubercles not only become much reduced in size, but are less congested and irritable. The mask I have used is one made by spreading zinc ointment on lint.

The beneficial effect of such a mask is undoubted, but why it should be good it is difficult to say; it may be that it protects the skin from cold, or it may be that the bacillus requires a certain amount of light to enable it to thrive, for it is to be noted that the microbe always grows more rapidly in those portions of the skin which are most exposed to light, such as the face, hands, and ankles.

Tonics, and in many cases stimulants, are needed to improve the tone of the system, and the anæmic condition to which the patients are reduced by the excessive discharges from ulcers must be combated by the use of iron, arsenic, and other appropriate remedies.

I have seen marked improvement follow the administration of a mixture containing pot. iodidi, hydrargyri, perchlor. pot. chlor. and decoct. sarsæ co. Salicylic acid also has a beneficial effect on the disease by its action on the cutis.

I have not found Chaulmoogra oil of much use in the treatment of leprosy, but, as other observers have been successful with it, I think it should be tried. All oils have a beneficial effect on the tubercles when they are well rubbed into the skin, but I think the good effect is mainly due to the rubbing, and not to the curative properties of the oil. In the Chaulmoogra-oil treatment the patient is subjected to a protracted course of massage, which must have a good effect on the tissues.

Nerve stretching has been tried with good results in anæsthetic leprosy. Here I think the *rationale* of the

treatment is obvious. The nerves are swollen and congested, the stretching to which they are subjected empties the blood-vessels and lymphatic spaces of the affected part, and thus relieves the congestion and improves the condition of the patient. It is probably for the same reason that the treatment by massage relieves a congested spot.

Leper patients are very partial to oils and ointments, with which they lubricate the skin, and which they rub into the unbroken surfaces, in some cases with good results. Ichthyol and Gurjun oil I have used without any good results. Iodoform ointment I have found the most useful of external applications. It not only acts beneficially upon the wounds, but, what is of more consequence in such a loathsome disease, it veils to some extent the fetor which arises from the wounds; this ointment is much appreciated by the patients.

Oakum makes an excellent dressing for the wounds, for it not only forms a soft pad, but is a good absorbent of the discharges, which are in some cases very copious.

The shallow ulcers which form on the lips of tubercular patients are exceedingly painful and sensitive, and should be protected by goldbeaters' skin, or some other suitable protective. All necrosed bones should be removed at once by operation. The patients bear operations well, and the wounds thus formed heal readily. Operations on the hands and feet of anæsthetic patients may be performed without anæsthetics, as the parts are more or less devoid of feeling.

I have found that tracheotomy and laryngotomy give patients great relief when the breathing has become oppressed through tuberculous infiltration of the larynx.

The tubercles which form on the conjunctiva may be removed from time to time with curved scissors, but eventually the eye becomes disorganised.

The eyes of anæsthetic patients should be protected artificially as much as possible from dust and other irritants, as the paralysed lids are of little use. All intercurrent affections should be treated in the usual manner.

As leprosy is a contagious and practically incurable disease, we can only hope to check its spread by adopting measures calculated to prevent infection.

The practical questions to be decided are:—Is it necessary to isolate all leper patients for life? If not, how far is the isolation of the diseased necessary, due regard being paid to the safety of the public?

All authorities are now agreed that the tubercular form of leprosy is the most dangerous to the public, for the discharges from the tubercular wounds teem with bacilli lepræ, and that anæsthetic leprosy is of little if any danger to the public, as the discharges are free from bacilli lepræ.

This being so, I am of opinion that all tubercular patients should be very strictly segregated during the ulcerative stages of the disease, but that before and after this period their isolation is not necessary.

I am further of opinion that it is unnecessary to segregate the anæsthetic patients, who should be allowed their liberty, or should be treated in the ordinary pauper wards of the country.

I would, however, suggest that proper records should be kept of each leper patient, whether in the asylums or not, and that the patients should all be periodically examined.

CHAPTER XVIII.

IS LEPROSY CURABLE?

THIN, on page 114 of his book on Leprosy, says, "With the disappearance of the spots, cessation of bullæ, and marked improvement in the general health, the patient may be said to be cured; atrophic and paralysed muscles and anæsthetic portions of skin being the only remnants of the disease." On page 228 of the same book, he says, "If a patient loses all the symptoms of tubercular leprosy, enjoys good health, but retains some slight symptoms of nerve leprosy of an unprogressive character, the case may be considered as much a case of cure as a case of phthisis in which all the symptoms have become arrested, although the patient is left with a patch of fibroid tissue in his lung, in which, doubtless, the spores of the tubercle bacillus are embedded." I am wholly in accord with Dr. Thin on these points, and am of opinion that leprosy is occasionally cured.

Many medical men, while virtually acknowledging the fact, cavil at the term used, and state that the patients are not cured, but that the disease is permanently arrested; the terms are, in my opinion, medically speaking, con-

vertible, for, if a disease is permanently arrested, most medical men would be content to pronounce it cured.

It is, however, a difficult matter to convince a sceptical mind that a cure has been effected in a patient, though one may be certain of the fact.

One observer goes so far, in speaking of leprosy, as to say that unless it can be proved by microscopical examination that no bacilli exist in any tissues of the body a case cannot be pronounced cured. Such a test is, however, impracticable, as it is obviously impossible to subject the tissues of a living person to a microscopical examination, but, regarding the matter from a reasonable and common-sense point of view, I think all must agree that anæsthetic leprosy is occasionally cured or permanently arrested. In these cases all the active signs and symptoms of the disease disappear, although the deformities may remain, and the patient, if not carried off by some intercurrent affection, will probably live to a ripe old age.

In anæsthetic leprosy the natural course of events is for the disease to expend itself, and if the patients could only stand the terrible battle for life they would all become cured; but unfortunately the strain is too great, and most of the patients die before the bacilli have been able to work out their own destruction.

In anæsthetic leprosy we know that the nerves are primarily affected or attacked, some say by the microbes themselves, but I think by the poison they secrete, and the destruction of the nerves gives rise to the terrible signs and symptoms of the disease.

The nerves at first become swollen and soft through interstitial inflammation, but inflammation of a chronic kind always leads to contraction and fibroid degeneration

of the tissues, so that the nerves become in time reduced to the condition of a fibrous band or filament, in which all traces of nerve-tissue are lost.

The sclerotic condition of this filament presents an effectual bar to any further growth of the bacilli, which, being deprived of all nourishment in this manner, die. It is in this way, I believe, that they work out their own destruction.

In tubercular leprosy the repeated attacks of congestion to which the tubercles are subjected in the course of the disease, produce, in like manner, a sort of fibroid degeneration of the tissues, and the more chronic the case is, the firmer and more fibroid the tissues become.

This fibroid degeneration of the tubercles causes them to become much reduced in size, and they become firmer and denser in consistency, and thus less able to support a mass of living organisms.

A recent tubercle is composed almost wholly of bacilli and their products; but an old tubercle is composed almost entirely of fibrous tissue.

This is the natural course of events in a tubercle when it becomes chronic, and I believe that if we could in any way expedite this fibroid degeneration of the tissues in them, we should do much to benefit the patients, if we did not actually cure the disease.

I believe we can bring about this much-to-be-desired result, and that if the disease is not too far advanced we can cure tubercular leprosy.

All observers have noticed that an acute attack of any disease which affects the skin, such as measles, small-pox, and erysipelas, has a most marked beneficial effect on tubercular leprosy. This, I believe, is entirely due to the

effects of the additional inflammation which is superimposed on the congestion natural to the disease. This intercurrent inflammation brings about sufficient fibroid degeneration of the tissues to bar the way to any further growth of the bacilli, if it does not actually cause their destruction.

The effects of an attack of erysipelas are so marked, not only in reducing the size of the tubercles, but also in diminishing their number, that I am forced to the conclusion that in this disease we have a means which, if properly and judiciously applied, must cure the tubercular form of leprosy (compare Plates III. and XXXV., also Plates IV. and XXXVI.).

The bacilli in an ordinary case of tubercular leprosy are for some years confined to the face and extremities. The early general symptoms of the disease being produced by the poison secreted by them, therefore if the microbes, which are at this time local, are destroyed, the general symptoms of the disease would soon disappear.

External applications are useless against them in the skin, and internal remedies are equally powerless to destroy them; but in these inflammatory affections we have a power to which we know the bacilli must succumb.

Of the inflammatory affections of the skin erysipelas is the most searching and therefore the most powerful. If one attack can produce the results shown in the two photographs (see Plates XXXV. and XXXVI.), then I think the fact is established that, in erysipelas we have a power which destroys the bacilli of leprosy, and once having this power, its application is only a matter of detail.

Erysipelas is always a dangerous disease, especially in a hospital, or in an asylum in which the patients have

broken surfaces, therefore I have not felt myself justified in putting my opinion on this matter to a practical test in the leper wards of the Robben Island Asylum, though many patients have expressed their willingness to undergo the ordeal. Many have contracted the disease spontaneously, and the part affected by erysipelas has in each case been much improved; unfortunately, however, the patients who are liable to contract erysipelas are those who have broken surfaces—that is, those who are in an advanced stage of the disease, and therefore the very patients to whom no lasting benefit could result from any treatment. For when once the tubercles extend to the mucous membranes the bacilli get beyond the reach of even erysipelas.

The two patients whose photographs I have produced were certainly two of the worst cases of tubercular leprosy in the asylum; but as may be seen even in them the erysipelas has effected a marked improvement. Compare the appearance presented by the patients before and after the attack of erysipelas (compare Plates III. and IV. with Plates XXXV. and XXXVI.).

If one attack of this can destroy some bacilli, as it undoubtedly has in the cases produced, then it is self-evident that more attacks must destroy all.

I believe one attack of facial erysipelas would destroy all the bacilli in the tubercles on that part of a person who has not had the disease for more than three years, a second attack would destroy the bacilli in the extremities if there were any located there; and thus I think by artificially producing two attacks of erysipelas in a recent case of tubercular leprosy the disease could be cured.

CHAPTER XIX.

SEGREGATION OF LEPERS.

In 1884 an act was passed making the segregation of leprosy compulsory in the Cape Colony, but for some reason it was not promulgated until 1892, when a large number of patients were sent to Robben Island.

The report of the Indian Leprosy Commission gave rise to some doubts as to the necessity of enforcing the segregation, and I raised the question of self-cured leprosy.

A commission was appointed by the Colonial Government to inquire into the matter, and to see what could be done to ameliorate the condition of the lepers.

The Commission was appointed in January, 1894, and sent in their final report during 1895.

After examining many witnesses, and as the result of information obtained, the Commission came to the following conclusions and made the following recommendations, which were presented to His Excellency the Governor of the Cape of Good Hope :—

General Conclusions and Recommendations.

1. Your Commissioners desire to affirm that leprosy is a source of danger to the health of the people of this

country. The facts adduced in this report show that the disease has steadily increased; and, moreover, the class of people mainly afflicted are those who, from their personal habits and hygienic surroundings, are likely to further the spread of the malady. The Commission are therefore of opinion that the existence and spread of leprosy call for such legislative action as will enable the Government to safeguard the interest of the whole community.

2. Your Commissioners are of opinion that leprosy is communicable from the diseased to the healthy, and that there is not sufficient evidence to show it is ever produced in any other way. The exact conditions under which contagion takes place, whether by inoculation only, or by such close contact as is indicated by dwelling in the same house, sleeping in the same bed, wearing the same apparel, eating out of the same utensils, using the same tools, &c., cannot at present be determined; but the evidence points undoubtedly to the fact that unfavourable hygienic conditions, such as overcrowding, uncleanly habits, unhealthy food, and poverty, strongly favour the communicability of the disease.

3. Your Commissioners are of opinion that leprosy is in some cases spontaneously arrested for longer or shorter periods, and in a very small proportion of cases it would appear that this arrest is permanent. There is no specific sign by which arrest can be recognised; but the healing of ulcers, the loss of limbs, a quiescent state of skin, a general appearance of good health, increase of weight, and the absence of any indication of active disease, external or internal, for a period of two years, may be regarded for all practical purposes as arrest of the disease.

4. Your Commissioners are of opinion that there is no

proof of the direct hereditary transmission of leprosy. They are further of opinion that an hereditary predisposition to the disease can only be adduced in so small a proportion of cases, that it cannot be regarded as having any important influence on its spread.

5. Your Commissioners are of opinion that conjugal intercourse may be permitted to lepers past the child-bearing age, but should be discouraged in every possible way between lepers at an earlier period of life. Conjugal intercourse between the leprous and healthy should not be permitted; but leper-villages or locations should be specially dealt with.

6. Your Commissioners have not obtained any proof regarding the duration of the period of incubation, and they are not disposed to make any recommendations regarding the "possibly infected."

7. Your Commissioners are of opinion that the compulsory segregation of persons suffering from leprosy is the only practical way of effectually arresting the spread of, and eradicating, the disease. They are of opinion that the present system of compulsory segregation should be modified in important respects. They would recommend that the degree of isolation necessary in each case of leprosy should be secured either in asylums, in licensed houses, in private dwellings, or in leper villages or locations. They are of opinion that one or more leper asylums should be erected on the mainland, and that unless such asylums are established the working of a compulsory segregation act will be rendered extremely difficult, and unnecessarily repugnant to the people. They recommend that regulations be drawn up prescribing the conditions under which patients should be

segregated, whether in asylums on the mainland, or elsewhere in private dwellings, in licensed houses, or in leper-villages or locations. Detailed recommendations in connection therewith will be found in the body of the report. The Commission would direct attention to the serious fact that a large number of cases of leprosy are at large in the native districts and territories, and that the disease is apparently on the increase in these areas.

8. Your Commissioners are of opinion that no methods other than segregation complete or modified exist, by which the spread of leprosy may be effectually arrested.

9. Your Commissioners are of opinion that in the event of leprosy being extinguished or diminished in this country, there is a danger of its being imported from other states or countries, and they would recommend that the disease be compulsorily notified in all bills of health.

10. Your Commissioners are of opinion that the notification of leprosy should be compulsorily enforced by legislation.

11. Your Commissioners are of opinion that no leper can be allowed, under any circumstances, to remain at large except under the conditions and regulations mentioned.

12. Your Commissioners are of opinion that all persons suffering from leprosy should be prohibited by law from following such occupations as would be attended with risk to the public.

13. Your Commissioners attach great importance to general measures of hygiene in dealing with the spread of leprosy, and they are of opinion that a vigorous enforcement of sanitary regulations would materially aid in

checking the spread of the disease. Efforts should be made throughout the whole colony to educate the people to the importance of hygiene and the dangers of contagion, and steps should be taken in the educational institutions of the colony to instruct pupils in the principles of health and sanitation.

14. Your Commission strongly affirms the necessity of a study of leprosy from its pathological and bacteriological aspects being inaugurated in South Africa, and considers that the medical officers of the leper institutions ought to have facilities granted to them for such study, and submits that, there being a bacteriological institute in the colony, arrangements should be made whereby the medical officers of the leper institutions may attend there for short periods, during which assistance and instruction in the use of modern methods of research may be given them by the director and staff of the institute mentioned.

CHAPTER XX.

PROPOSED MODIFICATION OF PRESENT SYSTEM OF SEGREGATION.

Synopsis.

The Commissioners, after carefully considering the subject of segregation of lepers, came to the following conclusions:—

1. There should be compulsory notification of every case of leprosy by the householder or occupier, and by the medical practitioner in attendance.

2. Isolation should be secured in asylums, in licensed houses, in private dwellings, or in leper-villages or locations. The class of patients to be admitted into these places of detention to be selected on certain broad lines.

3. One or more *asylums* for lepers should be erected on the mainland. Robben Island Asylum being used for pauper patients and convict lepers. *Licensed* houses should be conducted on the lines of the provision of the Lunacy Law of Scotland. *Private dwellings* would only be allowed to such patients as could satisfy a board that in every way segregation would be complete. *Leper-villages* and *locations* should be provided in the native territories.

4. No lepers should be allowed to follow occupations which would be attended with risk to the public, and no articles made by lepers should be allowed to be sold. Lepers should not be allowed to attend public meetings or public assemblies.

5. Laws affecting the segregation of lepers should be administered with the utmost tact and feeling, undue rigour defeats its own end. Enlist public sympathy. General hygienic measures most important in dealing with leprosy.

CHAPTER XXI.

REGULATIONS FRAMED BY GOVERNMENT FOR PRIVATE ISOLATION OF LEPERS.

EXTRACTS from Final Report of the Leprosy Commission, indicating the conditions and restrictions under which the Commission considers that the detention of lepers in private dwellings and licensed houses might be allowed.

Published by Authority.

The Commission lays down certain general principles by which the selection of cases for detention in licensed houses or private dwellings should be governed.

LICENSED HOUSES.

As regards detention in licensed houses the general principles may be summarised as follows:—

1. The consent of the Local Authority—*i.e.*, Municipal Council or Village Board—is required.

2. A licensed house would contain from two to four patients, except when the Municipal Council applies for a licence for more than four.

The licence could be withdrawn at any time.

3. Except in special cases, such as old married couples, each licensed house would receive patients of one sex only. The inmates would be subject also to regulations providing for :—

(1.) Regular medical inspection and report.
(2.) Supervision over conduct—*e.g.*, restrictions as to exercise, visits, disinfection, &c.—with a proviso that breach of the conditions would involve removal to the asylum on Robben Island.

Private Dwellings.

As regards isolation in private dwellings the conditions are very stringent.

The decision in such cases would be influenced by such considerations as the following :—

(*a*) The position of the dwelling, its relation to other houses, and its environment generally.

(*b*) The size and character of the dwelling, and the occupations and ages of the other dwellers therein. If the dwelling were used as a shop, bakery, laundry, &c., it would be manifestly unsuitable. A dwelling containing a number of young children would be looked on, *cæteris paribus*, unfavourably, and so on.

(*c*) The accommodation to be at the disposal of the patient. One separate room would be indispensable, and it should be completely detached from the dwelling-house, or at least have a door opening directly to the open air.

(*d*) The arrangements as to bedding, clothing, eating utensils, &c., also as to attendance. The bedding, clothing,

eating utensils, &c., would have to be entirely set apart for the use of the patient. Satisfactory arrangements would have to be made for the washing and disinfection of the clothes. Great cleanliness of the apartment and surroundings would be insisted upon, and the free use of disinfectants.

(*e*) The condition of the patient as to the stage of the disease. For example, whether in the very early stages or in the ulcerative stage.

(*f*) The conditions as to marriage and children. For example, if married, the age would be considered with special reference to child-bearing.

(*g*) Sanction would be withheld unless there was a reasonable expectation of the rules and regulations being adhered to; and therefore a degree of intelligence and trustworthiness would be essential in the applicant and his friends.

(*h*) The tenor of the report of the district surgeon or other approved medical practitioner, as to whether the necessary degree of isolation could be obtained would have great weight.

The patient would also be subject to regulations providing for:—

1. Periodical medical visitation and report at such times as may be directed.

2. Periodical visits by municipal or other authorised officer.

3. Periodical reports by relative or person in charge at such times as may be required.

4. Visits to patients; precautions to be taken; restrictions as to exercise, with a view to avoiding possible spread of disease, &c. &c.

All expenses would have to be borne by the lepers who are allowed the privilege of living in a private dwelling. Any breach of the conditions would involve removal to the Asylum on Robben Island.

COLONIAL SECRETARY'S OFFICE,
CAPE OF GOOD HOPE,
August, 1895.

PLATE I.

Mixed Leprosy of Three Years' Duration.

W. N. Admitted into the Robben Island Asylum in August, 1891, from Aliwal North. Aged at that time 16 years. Father, a Tambokie, alive and healthy. Mother, also a Tambokie, alive and healthy. No relative affected with leprosy.

Disease began in 1890 with contraction of left hand. In 1893 the face presented the appearance in the photograph. Small tubercles on eyebrow and chin; fingers of left hand contracted. No sores.

This case illustrates the formation of tubercles on the eyebrow.

PLATE I.

Mixed Leprosy of Three Years' Duration.

PLATE II.

TUBERCULAR LEPROSY OF THREE YEARS' DURATION.

J. K. Admitted into the Robben Island Asylum in November, 1892, from the Cape District. Aged 28 years. A Bastard. Farm labourer. Married. Wife and three children alive and healthy. No relative affected with leprosy.

Disease began in 1891 with red marks on the face.

This case illustrates the development of tubercles on the face.

PLATE II.

TUBERCULAR LEPROSY OF THREE YEARS' DURATION.

PLATE III.

Tubercular Leprosy of Seven Years' Duration.

C. A. Admitted into the Robben Island Asylum in October, 1888, from Darling. Aged at that time 25 years. Single. A groom. Father and mother, coloured, not affected with leprosy, dead. Three sisters alive and healthy, five brothers and two sisters dead, not of leprosy. No relatives affected with leprosy.

Disease began in 1887 with pimples on the face and swelling of the face. In 1893, when the photograph was taken, the face had become tuberculated, and the fingers had become swollen and œdematous. The patient had an ulcer on the left foot. Had during 1893 an attack of facial erysipelas, since which the tubercles on the face have become much reduced in size.

In 1894 several small shallow ulcers had formed on the left foot.

Mark the tubercles in all stages of development, some smooth and unbroken, others forming a pale summit, others ulcerated, and covered with hard scabs. Slight œdema of the eyelids.

Note also the soundness of the cutis in the proximity of the hair.

PLATE III.

TUBERCULAR LEPROSY OF SEVEN YEARS' DURATION.

PLATE IV.

Tubercular Leprosy of Seven Years' Duration.

P. C. Admitted into the Robben Island Asylum in March, 1892, from Clanwilliam. Aged at that time 20 years. Herd. Father, a coloured man, died, not a leper. Mother, a Hottentot, died, not a leper. Three sisters alive and healthy. No brothers. No relatives affected with leprosy.

Disease began in 1888 with swelling of the face and ulceration of the skin of the face. Photographed in 1893. Face much tuberculated, superficial ulcers on hands and feet. Right arm much swollen and also ulcerated. Right side of face very much swollen. Hands tuberculated. Fingers of left hand ulcerated and nails lost. Tubercles on soles of both feet. Tracheotomy performed to relieve breathing. An attack of facial erysipelas has now much reduced size and number of tubercles on the face.

Here note again the comparative freeness of the skin near the scalp from tubercles, and the tubercles on the ear. In many tubercles the pale summits are well shown.

PLATE IV.

TUBERCULAR LEPROSY OF SEVEN YEARS' DURATION.

PLATE V.

Tubercular Leprosy of Six Years' Duration.

V. S. Admitted into the Robben Island Asylum in December, 1893, from Namaqualand. Aged at that time 21 years. Single. Father, Dutch, not a leper, now dead. Mother, Dutch, alive and healthy. Two brothers alive and healthy, one brother died a leper. The whole family lived in one house. Two sisters alive and healthy. No other relatives affected with leprosy.

Disease began in 1889 with red spots on face. Photograph taken in 1893. Face swollen with numerous venules distinctly marked. Large ulcers on outer aspect of both legs.

Note the peculiar mottled appearance of the face, due to the congestion of the capillary network in the cutis.

PLATE V.

Tubercular Leprosy of Six Years' Duration.

PLATE VI.

Tubercular Leprosy of Six Years' Duration.

S. B. Admitted into the Robben Island Asylum in March, 1892, from the Orange Free State. Aged at that time six years. Father, a coloured man, living, a leper. Mother, died a leper.

Disease began in 1889 with swelling of the whole body. Maternal grandmother died a leper.

1893. Arms tuberculated to shoulders, and legs to knees. Eruption on body like that which would be produced by drops of water having fallen on the skin, and partially washed out the colour. Is suffering from scabies. Face much tuberculated.

Note the large, smooth, though well-defined, tubercles, and the thick lips so characteristic of this form of tubercular leprosy.

PLATE VI.

TUBERCULAR LEPROSY OF SIX YEARS' DURATION.

PLATE VII.

Tubercular Leprosy of Eight Years' Duration.

W. R. Admitted into the Robben Island Asylum in 1892, from Piquetberg. Aged at that time 46 years. Hottentot. Married. Wife and children alive and healthy. No relatives affected with leprosy.

Disease began in 1884 with drowsiness, epistaxis, and febrile symptoms, following by an eruption of red spots on the face.

1893. Face much tuberculated, covered with small, hard tubercles. Tubercles on hands and arms to elbows, and feet and legs to knees. Patient died after an acute attack of erysipelas.

Note the number of small, hard tubercles on the face, very characteristic of the third variety of tubercular leprosy.

PLATE VII.

TUBERCULAR LEPROSY OF EIGHT YEARS' DURATION.

PLATE VIII.

TUBERCULAR LEPROSY OF FIVE YEARS' DURATION. FOURTH VARIETY.

C. K. Admitted into the Robben Island Asylum in October, 1892, from Tulbagh. Aged at that time 15 years. Father, coloured, died, not a leper. Mother, coloured, alive and healthy. Four brothers and four sisters alive and healthy. Two brothers died, not lepers. No relatives affected with leprosy. Herd.

Disease began in 1890 with swelling of the face.

1893. Face and hands much swollon. No division into tubercles.

1894. Legs infiltrated to knees, and arms to elbows. Shallow ulcers forming on face.

Note the smooth condition of the skin, and the absence of distinct tubercles.

PLATE VIII.

TUBERCULAR LEPROSY OF FIVE YEARS' DURATION.

PLATE IX.

TUBERCULAR LEPROSY OF TWELVE YEARS' DURATION.

A. S. Admitted into the Robben Island Asylum in 1890, from Malmesbury. Aged at that time 27 years. Father and mother, Dutch, both dead, not lepers. Two brothers alive and healthy. No sisters. No relations affected with leprosy. Married. Wife and four children alive and healthy.

Disease began in 1882 with spots on forehead, and then over the whole body. Trunk mottled. A row of tubercles on each side of spine. Arms and legs tuberculated.

1894. Shallow ulcers on face. Mottling of trunk has now become papular.

Note the flattened tubercles on the arms conforming to some extent with the flexures of the skin, and showing the "islands" of the cutis.

PLATE IX.

TUBERCULAR LEPROSY OF TWELVE YEARS' DURATION.

PLATE X.

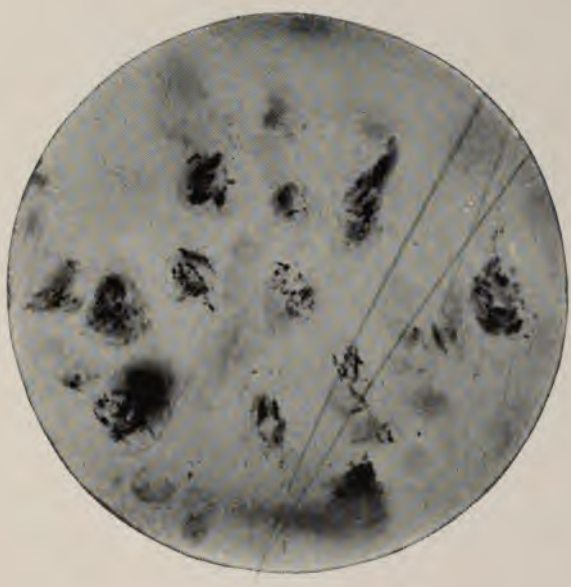
Tubercular Leprosy.

Microscopic photograph showing bacilli lepræ in the skin.

This photograph shows in a most excellent manner the way in which the bacilli are distributed in the tissues.

Note the masses of bacilli, forming dark somewhat circular patches, and the numerous smaller colonies of them, distributed all over the specimen. Note especially the enormous number of bacilli, which is always the case in the tubercles of leprosy.

PLATE X.



Bacilli in Tissues, × 750.
(*A. Pringle.*)

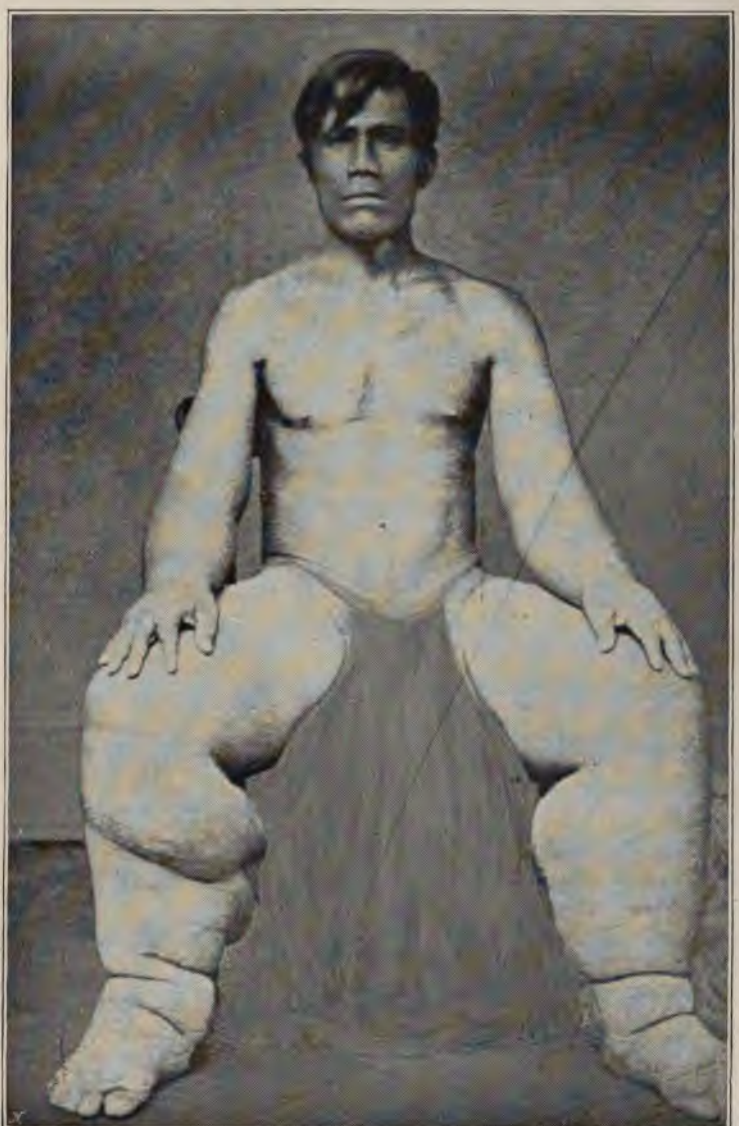
PLATE XI.

Elephantiasis Arabum.

Some writers still apply the old name of elephantiasis to leprosy. I have thought fit to produce a photograph of a patient suffering from elephantiasis Arabum in order to show the characters by which the disease may be distinguished.

Note the absence of disease in the face and on the trunk, the great enlargement of the extremities, especially the uniform swelling of the forearms and thighs, and the absence of tubercles. All these signs are sufficient to distinguish elephantiasis Arabum from tubercular leprosy.

PLATE XI.



ELEPHANTIASIS ARABUM.

PLATE XII.

Anæsthetic Leprosy of Four Years' Duration.

D. S. Admitted into the Robben Island Asylum in November, 1893, from Caledon. Aged at that time 12 years. Father and mother both Hottentots. No relatives affected with leprosy.

Disease began in 1891 with numbness of the hands.

Note the smooth face, devoid of any deformity, the contraction of the fingers, especially of fifth finger of left hand, also the commencing atrophy of the muscles of the forearm.

PLATE XII.

Anæsthetic Leprosy of Four Years' Duration.

PLATE XIII.

Anæsthetic Leprosy of Six Years' Duration.

J. K. Admitted into the Robben Island Asylum in November, 1893, from Fort Beaufort. Aged eight years. Herd. Father, a Hottentot, died, not a leper. Mother, a Hottentot, alive and healthy. Three brothers alive and healthy, one brother a leper. Two sisters died, not lepers. One paternal cousin a leper.

Disease began in 1889 with contraction of the right thumb.

1893. Fingers of both hands contracted. Left seventh nerve paralysed.

1894. Deep sinus in left foot; perforating ulcer on left foot and left hand.

1895. Fifth metatarsal bone removed for necrosis.

Note abbreviation and contraction of fingers, and perforating ulcer on the right index finger; note, also, anæsthetic patches on right cheek and forehead.

PLATE XIII.

Anæsthetic Leprosy of Six Years' Duration.

PLATE XIV.

ANÆSTHETIC LEPROSY OF FOUR YEARS' DURATION.

G. G. Admitted into the Robben Island Asylum in September, 1893, from Oudtshoorn. Aged 13 years. Father a Hottentot. Mother a black woman. Herd. No relatives affected with leprosy.

Disease began in 1891 with ulcers on the right knee.

1893. Fingers of both hands contracted, perforating ulcer, right heel, ulcer on right knee.

Note severe ulceration of the hands and emaciation of forearm and wrists.

PLATE XIV.

Anæsthetic Leprosy of Four Years' Duration.

PLATE XV.

ANÆSTHETIC LEPROSY OF SIX YEARS' DURATION.

D. M. Admitted into the Robben Island Asylum in February, 1893, from Port Elizabeth. Aged 13 years. Father died a leper. Mother, a Hottentot, died, not a leper. Two sisters alive and healthy, one sister a leper. Paternal grandmother a leper, one paternal uncle a leper.

Disease began in 1887 with pains in left arm and leg.

1893. Fingers and toes contracted, both seventh nerves paralysed. Perforating ulcer on left foot.

Note the paralysis of both facial nerves and deformity of left hand, the atrophy of the muscles of the hands, and the peculiar bend at the wrist joints.

PLATE XV.

Anæsthetic Leprosy of Six Years' Duration.

PLATE XVI.

Anæsthetic Leprosy of Ten Years' Duration.

G. J. Admitted into the Robben Island Asylum in July, 1889, from Fort Beaufort. Aged 20 years. Father and mother, Hottentots, both dead, not lepers. One brother alive and healthy, two brothers dead, not lepers. One sister dead, not a leper. Had two paternal cousins lepers.

Disease began in 1885 with ulcer on the right foot.

1893. Fingers and toes contracted, abbreviated, and tumefied. Paralysis of both seventh nerves.

1894. Perforating ulcer on right foot.

Note the contraction of the fingers, the nail of right ring finger resting on first phalangeal joint, also the white scars of bullæ on the fingers. Note also the paralysis of right buccal nerves.

PLATE XVI.

Anæsthetic Leprosy of Ten Years' Duration.

PLATE XVII.

ANÆSTHETIC LEPROSY OF SEVENTEEN YEARS' DURATION.

A. W. Admitted into the Robben Island Asylum in September, 1890, from Swellendam. Aged 30. Bastard. Labourer. No relatives affected with leprosy.

Disease began in 1878 with an ulcer under the left great toe. Had measles five years after leprosy began.

1890. Fingers and toes abbreviated and contracted. No sores.

Note the semi-contracted condition of the fingers of left hand, with contraction chiefly at the first phalangeal joint, the flatness of the back of the hand, and the atrophied appearance and condition of the muscles of the thumb. Note also the abbreviation at the last joint of the right index finger, and the amputation of the right little finger.

PLATE XVII.

Anæsthetic Leprosy of Seventeen Years' Duration.

PLATE XVIII.

Anæsthetic Leprosy of Ten Years' Duration.

G. T. Admitted into the Robben Island Asylum in 1889. A native of Natal. Father, a Zulu, dead, not a leper. Mother, also a Zulu, not a leper. Aged at time of admission 27 years. Had no brothers, but one sister, who is alive and healthy. No relatives affected with leprosy. Came into contact with lepers before he contracted the disease.

Disease began in 1884 with blisters on the hands.

Note the everted eyelids and the paralysis of left side of face. Note also the truncated fingers and the large white scar on the back of the right hand, due to a bulla.

PLATE XVIII.

Anæsthetic Leprosy of Ten Years' Duration.

PLATE XIX.

SYPHILITIC LEPROSY OF SIXTEEN YEARS' DURATION.

C. B. Admitted into the Robben Island Asylum in January 1892, from Murraysberg. Aged at that time 40 years. Father and mother Hottentots. Occupation, herd. No relatives affected with leprosy.

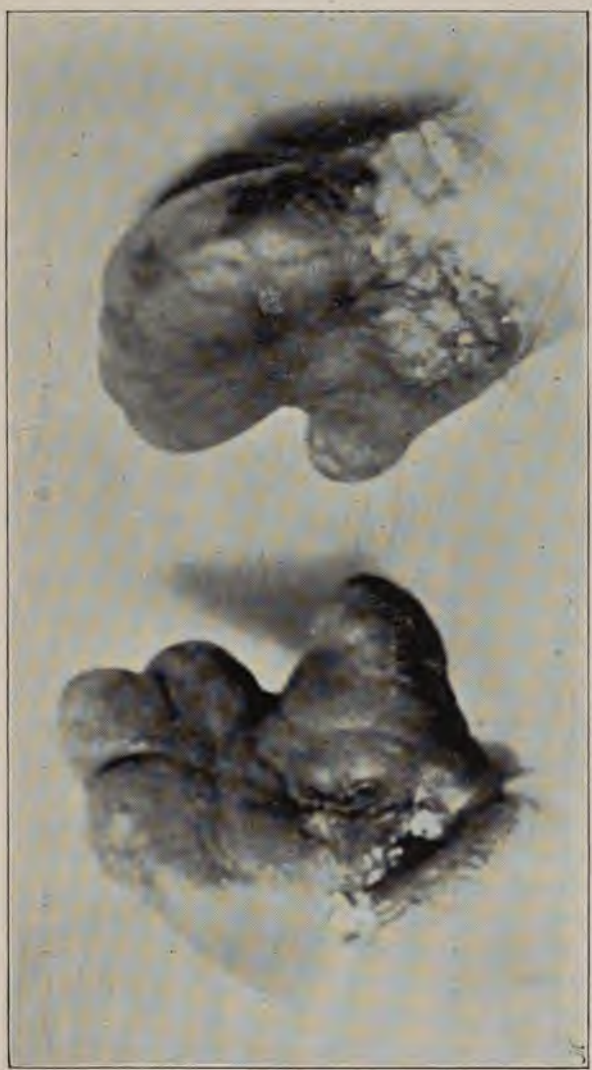
Disease began in 1878 with an abscess on the left index finger.

1893. Fingers and toes truncated. Both seventh nerves paralysed.

1894. Ulcer on right hand and on right foot.

Note the truncation of all the fingers, the great tumefaction of the stumps, the large ragged ulcer on right stump, and the scars on the left wrist.

PLATE XIX.



Syphilitic Leprosy of Sixteen Years' Duration.

PLATE XX.

Anæsthetic Leprosy of Twenty-three Years' Duration.

C. I. Admitted into the Robben Island Asylum in March, 1892, from King Williamstown. Aged at that time 35 years. Father and mother both Hottentots. Father alive and healthy. Mother died, not a leper. Single. Occupation, farm labourer. The patient cohabited with a leper woman before the disease manifested itself in him.

The disease began in 1872 with a feeling of cold and heat, alternating in the index finger of the left hand. Four months after this there was an eruption of yellow spots on the body, and blisters formed on the hands, arms, and legs; after which the fingers became contracted. The lower third of the bones of the forearm and legs were subsequently absorbed without the formation of ulcers.

The patient had seven nephews, of whom three were lepers, the other four are alive and healthy. Has one brother alive and healthy, and has had three sisters, not lepers.

In 1893, when the photograph was taken, the hands and feet had become flail-like, both seventh nerves were paralysed, and there was extensive leucoma of the right cornea. Has no sores, but has a severe cough, with blood-stained expectoration.

Note the flail-like appearance of the hands, due to absorption of the bones without ulceration of the skin.

PLATE XX.

Anæsthetic Leprosy of Twenty-three Years' Duration.

PLATE XXI.

Anæsthetic Leprosy of Seven Years' Duration.

K. S. Admitted into the Robben Island Asylum in January, 1893, from the Cape District. Aged at that time 22 years. Father, a coloured man, alive and healthy. Mother, also coloured, dead, not a leper. Six brothers alive and healthy. One brother a leper. Four sisters alive and healthy. No other relatives affected with leprosy. Former occupation, farm labourer.

Disease began in 1888 with an ulcer under the great toe of the left foot.

1893. Fingers abbreviated and contracted. Both seventh nerves paralysed. Perforating ulcer under sole of left foot.

This photograph shows the staring eye due to partial absorption of the lower eyelids, so characteristic of leprosy.

PLATE XXI.

ANÆSTHETIC LEPROSY OF SEVEN YEARS' DURATION.

PLATE XXII.

Anæsthetic Leprosy of Ten Years' Duration.

K. C. Admitted into the Robben Island Asylum in November, 1894, from Caledon. Aged at that time 20 years. Father, a European, alive and healthy. Mother, a European, died a leper. Two brothers alive and healthy. One brother died a leper.

The history of the disease, as it affected the family of this patient, is interesting. The first to become a leper was a maternal aunt of the patient, then the mother of the patient, then his eldest brother, and lastly the patient himself. The father, though cohabiting with a diseased wife until her death, escaped the disease. The patient lived with his diseased mother and brother from infancy.

Note the miserable look of the patient, due to the paralysis of the muscles of expression.

PLATE XXII.

Anæsthetic Leprosy of Ten Years' Duration.

PLATE XXIII.

Anæsthetic Leprosy of Four Years' Duration.

M. W. Admitted into the Robben Island Infirmary in August, 1892, from Cape Town. Aged at that time 25 years. Father, an American negro, dead, not a leper. Mother, a Hottentot, dead, not a leper. Is married, but has no children. Husband alive and healthy. No relatives affected with leprosy. Former occupation, cook and laundress.

Disease began in 1891 with pains in the feet, followed by ulceration of the feet.

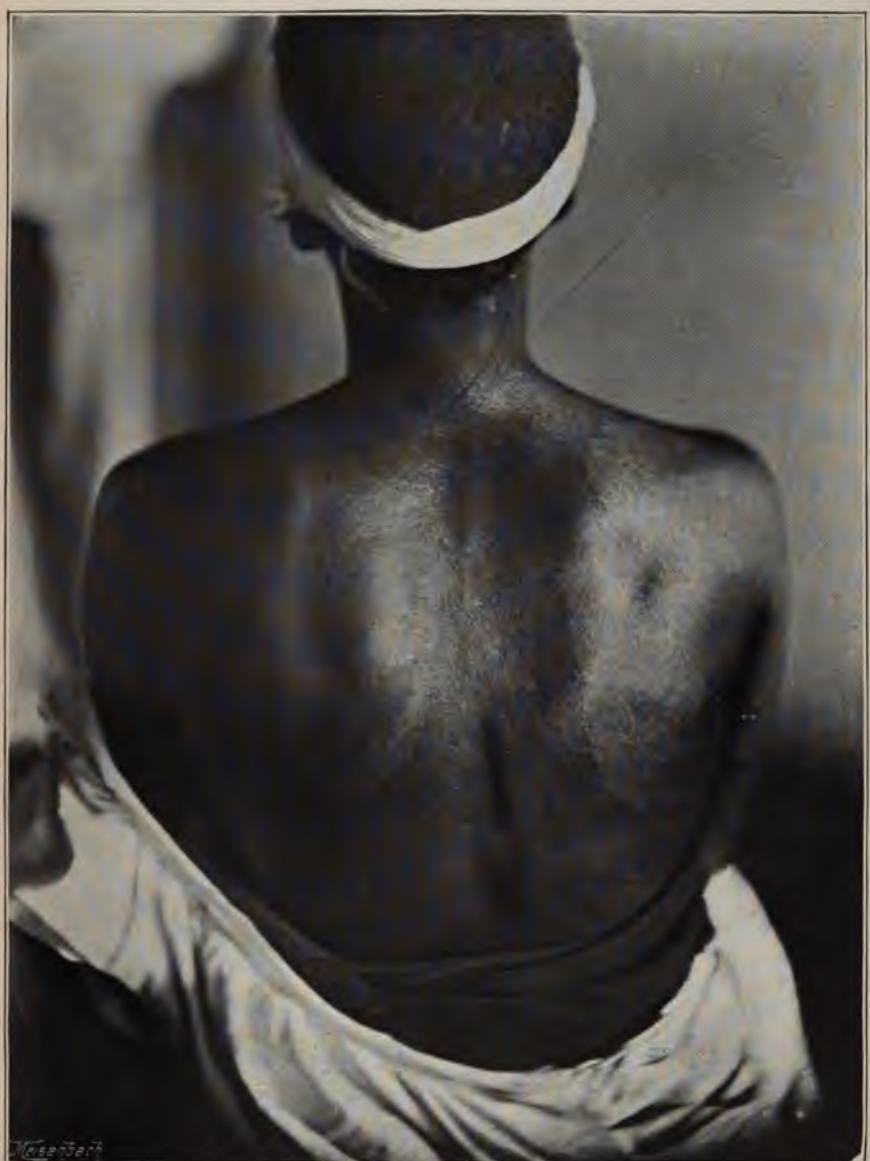
1893. Fifth fingers of both hands contracted. Perforating ulcers on both heels. Fifth toes of both feet gone. Numerous anæsthetic patches on the back, edges of which are slightly raised and scurvy.

1894. Margins of anæsthetic patches have now become lighter in colour and less defined. Severe pains in the chest, with cough.

1895. Severe and recurrent hæmoptysis.

Note the white anæsthetic patches on both shoulders, most marked on the right side. The patches on this patient are, as will be seen, unusually white, and in the photograph are almost indistinguishable from the high lights.

PLATE XXIII.



ANÆSTHETIC LEPROSY OF FOUR YEARS' DURATION.

PLATE XXIV.

Anæsthetic Leprosy of Ten Years' Duration.

S. I. Admitted into the Robben Island Asylum in July, 1892, from Kimberley. At that time 22 years of age. Father a German. Mother a Colonial. Both alive and healthy. Has one brother a leper. No other relatives affected. Is single, and a laundress.

Disease began in 1885 with numbness of the fingers, first of the right hand, then of the left, the fingers subsequently became contracted. Both feet infiltrated. Perforating ulcer under first toe of right foot. Both seventh nerves paralysed, left most affected. Numerous anæsthetic patches on trunk.

The patch shown on the plate is unusually distinct. It is, moreover, much depressed on account of the atrophy of the tissues. In the spot may be seen a small island of fairly healthy tissues. This island will, in all probability, enlarge at the circumference until the patch becomes healthy again.

PLATE XXIV.

ANÆSTHETIC LEPROSY OF TEN YEARS' DURATION.

PLATE XXV.

Mixed Leprosy of Seven Years' Duration.

P. G. Admitted into the Robben Island Asylum in May, 1892. Aged at that time 12 years. Father and mother, Hottentots, alive and healthy. One sister alive and healthy. One paternal uncle and one paternal aunt died lepers. One cousin a leper. All the other members of a large family healthy.

Disease began in 1887 with contraction of fingers.

1893. Fingers semi-contracted and numb. No ulceration.

Note the light patches on right cheek, on left cheek, above left eyebrow, on the chin, and on both the hands.

PLATE XXV.

MIXED LEPROSY OF SEVEN YEARS' DURATION.

PLATE XXVI.

Mixed Leprosy of Ten Years' Duration.

W. M. Admitted into the Robben Island Asylum, from Albany, in February, 1892. Aged at that time 45 years. A gardener by occupation. Married. Wife and children all alive and healthy. Father and mother Fingoes, not lepers.

Disease began in 1885 with pain in left arm.

1893. Face tuberculated. Fingers and toes abbreviated and contracted. No sores.

1894. Had an attack of pleurisy.

Note the well-developed tubercles on the face with partial paralysis of the lower eyelids, also the puffiness of the hands, especially the right, at the wrists, with semi-contraction of the small and ring fingers of right hand, and also the general thickening of the fingers. On the outer aspect of the little finger of the right hand is a scar, the result of a burn.

PLATE XXVI.

Mixed Leprosy of Ten Years' Duration.

PLATE XXVII.

Mixed Leprosy of Eleven Years' Duration.

M. K. Admitted into the Robben Island Asylum in April, 1887, from Cape Town. Aged at that time 32 years. Father, a Mozambique, alive and healthy. Mother, a Malay, died, not a leper. Three brothers and four sisters alive and healthy. Married. Wife and two children alive and healthy. No relative affected with the disease. Former occupation, coolie; working at Cape Town Docks.

Disease began in 1883, after exposure to severe cold and wet, with pains in the soles of the feet and palms of the hands, followed in 1894 with contraction of the fingers.

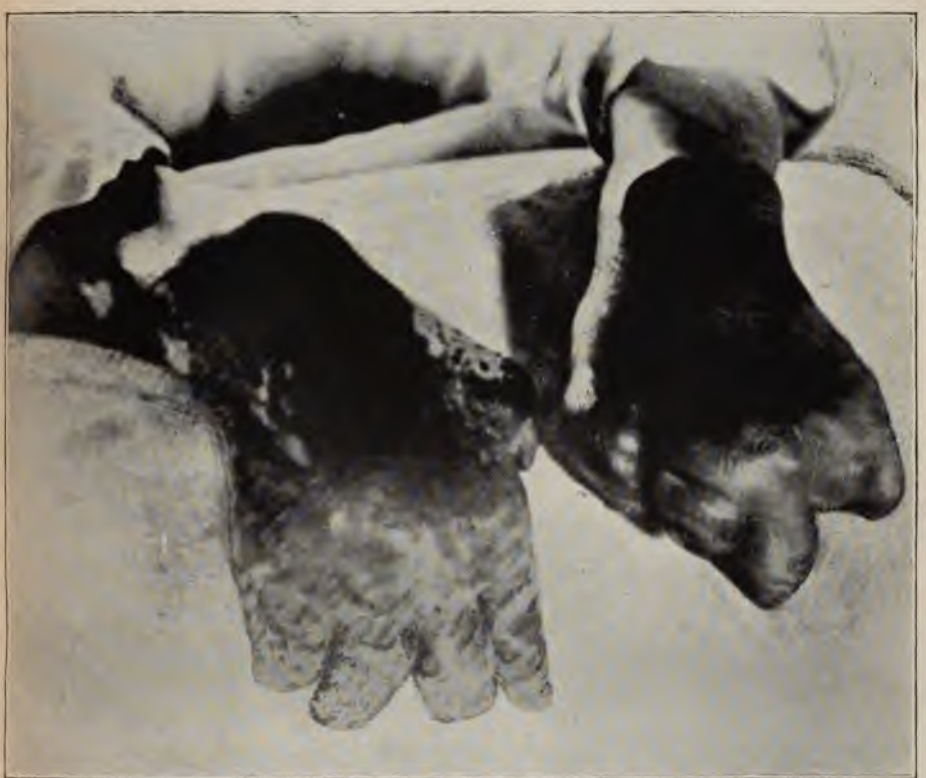
1893. Fingers and toes contracted and abbreviated; both seventh nerves affected. Had one attack of facial erysipelas.

1894. Face tuberculated and ulcerated, numerous shallow ulcers on both legs, perforating ulcer on both feet.

1895. Superficial ulcers on face, œdema of feet and legs. Urine albuminous. Dropsy. Death.

Note the mottled appearance of the hands due to the white patches, resulting from bullæ and burns. Note also the contraction, tumefaction, and abbreviation of the fingers.

PLATE XXVII.



MIXED LEPROSY OF ELEVEN YEARS' DURATION.

PLATE XXVIII.

Mixed Leprosy of Twenty-five Years' Duration.

A. K. Admitted into the Robben Island Asylum in September, 1892, from Clanwilliam. Aged at that time 52 years. Occupation, farm labourer.

Disease began in 1870, after the patient had "caught cold by going into the water whilst heated." The first symptoms were a loss of sensation in the left thumb, and a feeling of pins and needles in the arms and legs. The patient lived for six years with a sister who was a leper.

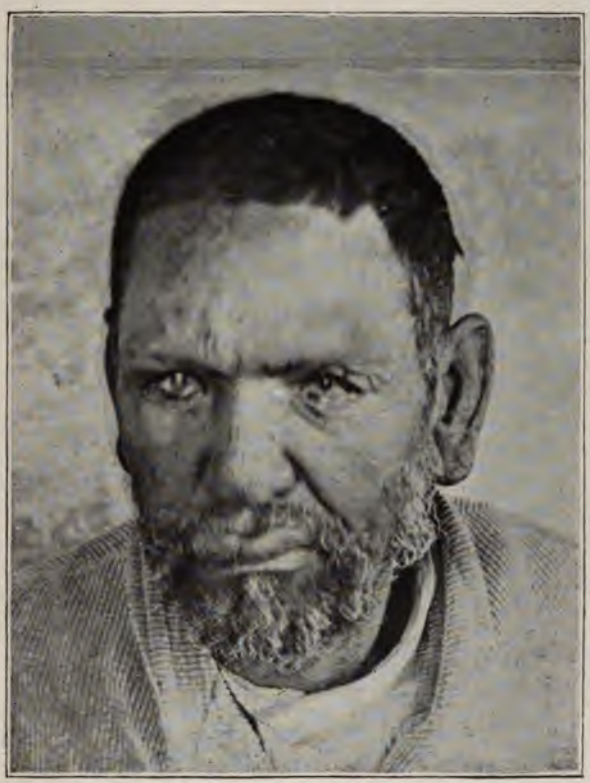
The history of the disease in the family of this patient is interesting. The patient had two paternal aunts, one of whom was a leper—the first in the family. The next to contract the disease was the youngest sister of the patient, the three eldest sisters escaping; then the patient's father contracted the disease, and lastly the patient himself. Four sons of the leprous aunt became lepers, but at what period of the history they contracted the disease I was unable to elicit.

The patient had four sisters, one of whom became a leper and died childless; one sister, herself healthy, had four children, one of whom became a leper. Another sister, also healthy, had three children, all lepers; the fourth sister was healthy and had no children.

1893. Fingers and toes contracted and truncated. Both seventh nerves paralysed. Shallow ulcers on face. Both conjunctivæ invaded by tubercles.

Note the tubercles in the eyes. These tubercles are soft and congested. Note also the paralysis of the right side of the face, the flaccid upper lip, and the thickening of the skin of the face, especially on the forehead.

PLATE XXVIII.



MIXED LEPROSY OF TWENTY-FIVE YEARS' DURATION.

PLATE XXIX.

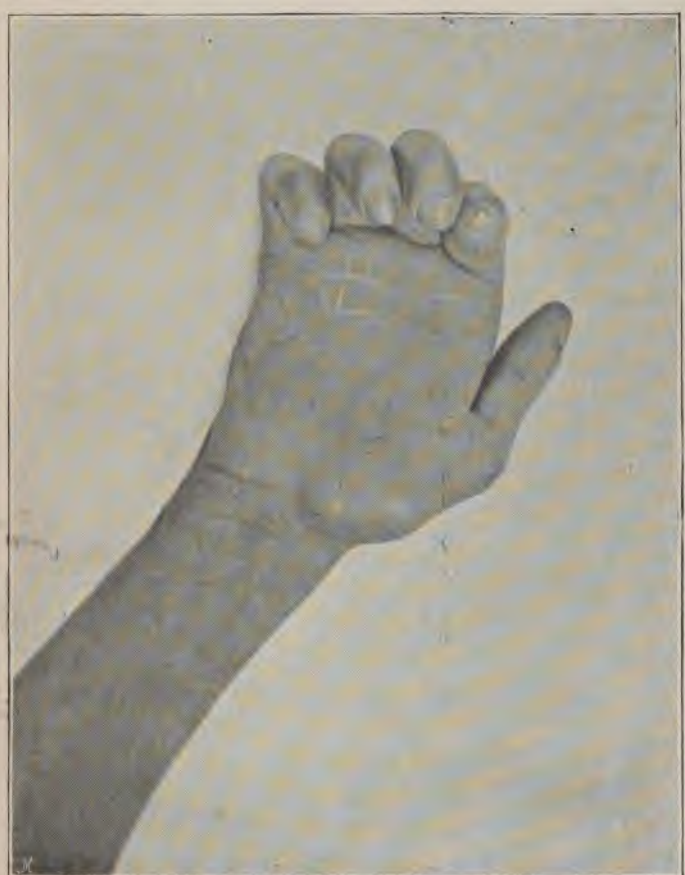
Anæsthetic Leprosy of Four Years' Duration.

M. K. Admitted into the Old Somerset Hospital in June, 1895, from Cape Town. Aged at that time 60 years. Mason by trade. Widower. Father and mother, Malays, both dead, not lepers. Wife died, not a leper. Two sons alive and healthy. Two daughters alive and healthy. No relatives affected with leprosy.

Disease began in 1891. The patient was working on a scaffold, from which he fell and hurt his knees. He bound ligatures round both his legs above the knees and "stopped the circulation of the blood." Shortly after this accident his hands became paralysed. The fingers became contracted; but he has not had any ulceration except those due to burns. Eyes are now affected; large ulcer on right heel, due to blistering of the feet from walking.

Note the "main en griffe," the flatness of the hand, and absorption of the muscles of the hand from atrophy. Note especially the manner in which the fingers are flexed at the second joint; also the atrophy of the muscles of the forearm.

PLATE XXIX.



ANÆSTHETIC LEPROSY OF FOUR YEARS' DURATION.

PLATE XXX.

Syphilitic Leprosy of Fifteen Years' Duration.

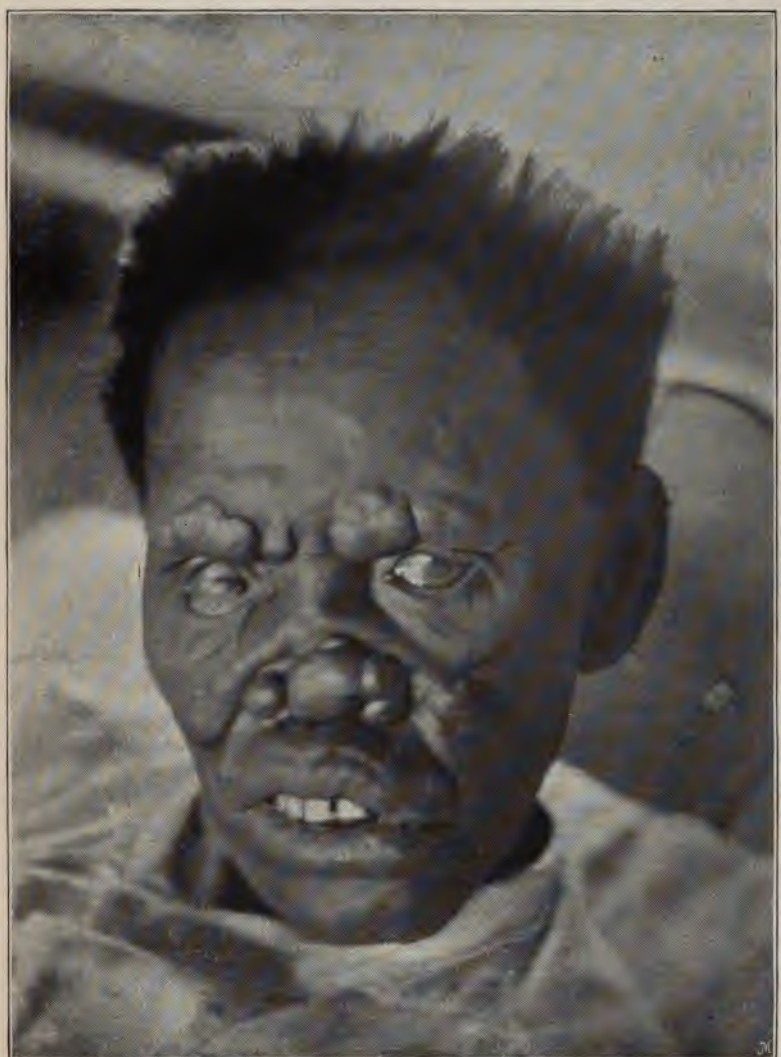
B. Admitted into the Robben Island Asylum in September, 1890, from Simonstown. Aged at that time 40 years. Single. Herd. No relatives affected with leprosy.

Disease began in 1880 with swelling of the face, followed by an eruption of yellow spots all over the body.

1893. Face tuberculated, fingers and toes contracted and tumefied; numerous ulcers on the trunk.

Note the tubercles on the face and in the conjunctiva, the sunken nose, and paralysis of the left side of the face.

PLATE XXX.



SYPHILITIC LEPROSY OF FIFTEEN YEARS' DURATION.

PLATE XXXI.

YPHILITIC LEPROSY OF TEN YEARS' DURATION.

S. Admitted into the Robben Island Asylum in 1890, from Queenstown. Aged at that time 56 years. Father a Scotchman. Mother a Hottentot. Both healthy. Married. Wife and two children alive and healthy. Former occupation, an evangelist.

Disease began in 1883 with spots on the extremities, and numbness of hands and feet.

1893. Fingers abbreviated, contracted, and tumefied; the toes in the same condition. Face infiltrated, and covered with shallow ulcers and scars; nose gone; both seventh nerves paralysed; body much emaciated. Pleurisy. Cough. Death.

Note size of eyes, due to contraction of cicatricial tissues, the ragged appearance of cutis, and the absence of well-defined tubercles.

PLATE XXXI.

Syphilitic Leprosy of Ten Years' Duration.

PLATE XXXII.

SYPHILITIC LEPROSY OF THE MIXED FORM OF SIX YEARS' DURATION.

J. A. Admitted into the Robben Island Asylum in February, 1892, from the Paarl. Aged at that time 14 years. Father, a coloured man, not a leper, dead. Mother, also a coloured woman, died a leper. Four brothers and one sister alive and healthy. No other relatives affected with leprosy. Patient lived with, and took care of, his mother for four years whilst she was suffering from the disease.

Disease began in 1887 with ulcers on the legs.

1893. Fingers of both hands contracted. Right hand tumefied. Face tuberculated.

Note the absence of hairs on eyebrows, irregular thickening of cheeks, paralysis of lower eyelids, most marked on left side; also thickening of cutis of the face, especially of the cheeks, and the scar of an abscess in the neck.

PLATE XXXII.

SYPHILITIC LEPROSY OF SIX YEARS' DURATION.

PLATE XXXIII.

SYPHILITIC LEPROSY OF THE MIXED FORM OF EIGHT YEARS' DURATION.

J. K. Admitted into the Robben Island Asylum in March, 1892, from Simonstown. Aged at that time 17 years. Father and mother, coloured, both alive and healthy. Five brothers alive and healthy, two brothers dead, not lepers. One brother a leper. Two sisters dead, not lepers. One sister alive, a leper. Paternal grandmother died a leper. The former occupation of the patient was that of a herd. Patient resided with his sister, who was a leper from infancy, but the disease only developed after he had caught a severe cold.

Disease began in 1887 with the loss of sensation in legs and arms, and with spots on face. The elder sister first contracted the disease, then the patient, and lastly his younger brother.

1893. Face tuberculated, both seventh nerves paralysed.

1894. Numerous shallow ulcers on face, these soon cicatrised. Large shallow ulcers on lower third of outer aspect of left leg, another on lower half of right leg. Breathing laryngeal.

Note the thickened eyebrows, the scars on right cheek, on chin, below left eye, and above left eyebrow, the ulceration of nose, the partial loss of nasal cartilage, the retraction of upper lips, and the ill-shaped muddy eyes.

PLATE XXXIII.

SYPHILITIC LEPROSY OF EIGHT YEARS' DURATION.

PLATE XXXIV.

Syphilitic Leprosy of Fifteen Years' Duration.

E. Admitted into the Robben Island Infirmary in August, 1892, from Fort Beaufort. Aged at that time 25 years. Single. Father, a coloured man, alive, and healthy. Mother, a Hottentot, died, not a leper. Three brothers alive and healthy, and five sisters alive and healthy. No relatives affected with leprosy.

Disease began in 1878 with the development of yellow spots all over the skin, followed by blisters, and shortly afterwards by contraction of the fingers, which also became rigid.

In 1890 the patient became blind through ulceration of the eyes.

In 1893, when the photograph was taken, the hands of the patient had become truncated, and the feet had become, as may be seen in the photograph, a shapeless mass; numerous ulcers had also formed on the extremities. The scalp was infiltrated and the hair removed in large patches. The patient died in the same year from general prostration.

Note the absence of the toes and metatarsal bones, the ragged appearance, especially of the right foot, and the white patches of cicatricial tissue.

PLATE XXXIV.

Syphilitic Leprosy of Fifteen Years' Duration.

PLATE XXXV.

TUBERCULAR LEPROSY OF SEVEN YEARS' DURATION AFTER ERYSIPELAS.

Compare this plate with Plate III., where the condition of the patient is shown before the attack of erysipelas.

Note the general improvement in the appearance of the cutis, especially of the cheeks, nose, and lower part of the face. All the tubercles have diminished in size, and many have completely disappeared.

Note especially that the most marked improvement is in those portions where the tubercles are the most recent.

PLATE XXXV.

TUBERCULAR LEPROSY AFTER ERYSIPELAS.

PLATE XXXVI.

TUBERCULAR LEPROSY OF SEVEN YEARS' DURATION AFTER ERYSIPELAS.

Compare this plate with Plate IV., where the condition of the patient before the attack of erysipelas is well shown.

Note the very marked improvement in the appearance of the face, and the general subsidence of the tubercles—in parts they seem to have disappeared entirely, while in other parts they are much reduced in size. Note especially the great reduction of the swelling about the right eye.

PLATE XXXVI.

TUBERCULAR LEPROSY AFTER ERYSIPELAS.

PLATE XXXVII.

KELIS.

This patient was sent to the asylum as a leper, but the size and elevation of the swelling, which is greatest on the left cheek; the peculiarly soft feeling it presented on the surface, and the hard, fibrous base; the absence of congestion, and the unsymmetrical appearance of the tumour, were sufficient to distinguish it from tubercular leprosy.

PLATE XXXVII.

KELIS.

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